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A Comparison of Pressure Pain Detection Thresholds in Chronic Low Back Pain Sufferers and Pain Free Volunteers

by

Ronald Jason Scott Giesbrecht

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the

requirements for the degree of Master of Science

in

Department of *Physical Therapy*

Edmonton, Alberta

Fall 2003

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Degree: Master of Science

Year this Degree Granted: *2003*

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Faculty of Graduate Studies and Research

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "**A Comparison of Pressure Pain Detection Thresholds in Chronic Low Back Pain Sufferers and Pain Free Volunteers**" submitted by Ronald Jason Scott Giesbrecht in partial fulfillment of the requirements for the degree of Master of Science.

Date Approved by Committee: Aug 15, 2003

A Comparison of Pressure Pain Detection Thresholds in Chronic Low Back Pain Sufferers and Pain Free Volunteers

Abstract

Low back pain is a major source of disability and economic burden in western society. Despite the prevalence of low back pain and the tremendous costs associated with it, assessment techniques for determining the etiology and severity of low back pain are less than ideal. The interpretation of pain or tenderness reported during the manual palpation of a low back pain sufferer is complex and often misleading. Alterations in tissue sensitivity may occur as the nervous system adapts in response to neurobiological and biopsychosocial influences. The purpose of this research project was to compare the pressure pain detection threshold of chronic and acute low back pain sufferers and healthy pain free subjects. Thirty subjects seeking health care for chronic low back pain were recruited from the offices of health professionals and compared for differences in pressure pain detection threshold (PPDT) at six test sites, examined bilaterally, with thirty pain free volunteers. Due to difficulties with recruitment, the acute low back pain group was excluded from the study. The results demonstrated that chronic low back pain sufferers tend to show global decreases in pressure pain detection threshold compared to pain free subjects. Chronic low back pain sufferers showed the most significant decreases in pressure pain detection threshold at the low back region. It is suggested that neurobiological and/or biopsychosocial influences may have contributed to the alterations in pressure pain detection threshold evident in chronic low back pain sufferers.

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The Problem

Low back pain is a major source of disability and economic burden in western society. It is one of the most common reasons for disability in persons under the age of 45 and the most frequent symptomatic reason for visiting a physician's office and work absenteeism.[1,2] Epidemiological research reports a 60-80% lifetime prevalence of low back pain in the industrialized world.[3,4] Several investigations have found that 90-95% of low back pain sufferers will recover and return to work within 6-12 weeks.[5,6] Yet 5-10% of low back pain sufferers report persistent pain and disability after three months and account for 75-95% of the costs related to low back pain.[7] A small proportion of chronic low back pain sufferers consume a significantly disproportionate amount of the financial and clinical resources. Abenhaim et al (1988) observed that nearly one-third of compensation costs for occupational accidents are directly related to back pain and 70% of compensation costs for back pain are accounted for by only 7% of cases.[8] In 1998, a review of occupational low back pain claims in the US determined that low back pain sufferers comprise 20% of total injury claims yet consumed 60% of the total costs associated with all injury claims.[9] Frymoyer et al (1991) estimated the direct and indirect costs of low back pain in the United States (US) at $50-100 billion annually making it the most expensive industrial injury in the US.[3] In a more recent study, Maniadakis and Gray (2000) investigated the economic burden of back pain in the United Kingdom through cost of illness analysis.[10] Their analysis combined direct and indirect costs and provided a baseline estimate on the costs of low back pain at between £6650 and £12300 million ($10.6-$19.7 billion USD). The authors suggest that back pain imposes a greater economic burden in the UK than any other known disease.

Despite the prevalence of low back pain and the tremendous costs associated with it, assessment techniques for determining the etiology and severity of low back pain are less than ideal. Traditionally, the clinical assessment of low back pain relies on a comprehensive physical examination often involving the evaluation of tenderness. Tenderness is typically assessed by the clinician with manual palpation. Manual palpation is a simple test for pain provocation accomplished by pressing on painful and pain free tissues to determine the location of irritable tissue and the severity of tissue damage. The interpretation of pain or tenderness reported during the manual palpation of a low back pain sufferer is complex and often misleading.

The nociceptive system is not composed of hardwired pain pathways; rather it constantly changes, operating in different states of sensitivity or excitability depending on the immediate need and the dominant pain mechanism activated by the central nervous system.[11] Manual pain provocation is not always directly proportional to the severity of tissue damage and does not necessarily imply damage to tissues beneath the examiner's fingers. Alterations in tissue sensitivity may occur as the nervous system adapts in response to neurophysiological and biopsychosocial influences. As a result, pain may persist long after the tissue is healed and global changes in pain threshold and hypersensitivity may ensue in healthy tissues distal to the low back or related neuroanatomical regions. Pain complaints in unrelated body regions and spontaneous

exaggerated pain outside the neuroanatomical sensory distribution of the suspected lesion may follow sensory threshold changes. This shift in pain processing may be responsible for ongoing pain instead of persistent tissue injury or inflammation. Numerous studies have demonstrated the development of mechanical hypersensitivity and persistent pain in animals and humans.[12,13,14,15] The evaluation of tenderness without considering the possibility of local and global hypersensitivity due to physiological or biopsychosocial influences may lead the clinician to misinterpret clinical findings obtained during the physical exam of a low back pain patient.

Purpose

The purpose of this research project was to compare the pressure pain detection threshold of acute and chronic low back pain sufferers and healthy pain free subjects. This comparison was designed to identify whether differences in sensitivity exist in low back pain sufferers as compared to pain free control subjects and whether differences occur between subjects reporting acute and chronic low back pain. Changes in the distribution of mechanical sensitivity between those with acute and chronic low back pain may point towards a relationship between pain duration and pressure pain detection threshold (PPDT). In addition, evidence obtained from pressure pain detection threshold measurements in acute and chronic low back pain subjects compared to pain free volunteers may help clinicians to understand the patterns of pain presentation in low back pain patients.

Research Questions

1. Do regional and global differences in PPDT exist between chronic low back pain, acute low back pain, and pain free subjects?
2. Do chronic low back pain subjects demonstrate increased sensitivity (lower PPDT score) at the middle phalanx of the second finger compared to acute low back pain sufferers and pain free subjects?
3. Is there an association between global PPDT and pain drawing score?

Hypotheses

1. <u>Primary Hypotheses</u>:
    a. *Chronic* low back pain sufferers will demonstrate a lower global PPDT compared to *acute* low back pain subjects and pain free volunteers.
        i. Null Hypothesis: No difference in pain threshold exists, at the distal test sites, between *chronic* low back pain sufferers, *acute* low back pain sufferers, and pain free subjects.
    b. *Chronic* and *acute* low back pain sufferers will demonstrate a lower PPDT at sites related anatomically to the low back region compared to pain free subjects.
        i. Null Hypothesis: No difference in pain threshold exists, at sites related anatomically to the low back region, between *chronic*

low back pain sufferers, *acute* low back pain sufferers, and pain free subjects.

c. *Chronic* low back pain sufferers will demonstrate a lower pain threshold at sites unrelated to the low back region compared to *acute* low back pain sufferers, and pain free subjects.
    i. Null Hypothesis: No difference in pain threshold exists, at sites unrelated to the low back region, between *chronic* low back pain sufferers, *acute* low back pain sufferers, and pain free subjects.

2. Secondary Hypothesis:
    a. Higher Pain Drawing scores will be associated with lower global PPDT scores in low back pain subjects.
        i. Null Hypothesis: No correlation will exist between pain drawing score and global pain threshold in low back pain subjects.

Background and Literature Review

Low Back Pain

Acute Low Back Pain

Acute low back pain has been arbitrarily defined as low back pain present for three weeks or less.[16] It has been estimated that 75-90% of adults experience acute low back pain at some point.[17] Von Korff and Saunders (1996) estimate that nearly 90% or acute low back pain sufferers with associated work absence return to work in less than 12 weeks but up to 50% experience a recurrence of symptoms and ongoing functional limitations.[18] Researchers estimate that more than 50% of first time acute low back pain sufferers will experience a second episode of pain within a year and over half of them will report persistent low back pain 6 to 12 months later.[7,19] The majority of acute low back pain sufferers will either recover significantly in two months or risk developing chronic low back pain.[20] About 1% of persons reporting acute low back pain will develop ongoing persistent symptoms extending beyond the normal time frame for tissue healing.[21]

Acute low back pain sufferers tend to report all or some of the following symptoms: intermittent or constant back ache, pain with movement, pain worsening with activity, pain increasing with sustained postures, and latent nerve root pain.[22] Pain may be reported at the center or the right, left or both sides. Unilateral pain typically radiates no farther than the gluteal fold, with no neurologic signs.[23] In some instances, symptoms (pain, paresthesias) may extend along the distribution of a lumbar nerve root dermatome, with or without neurologic signs. Bilateral leg pain may be reported but is much less common.

The Underlying Pathology of Acute Low Back Pain

Tissue injury in the low back region initiates noxious stimulation to activate nociceptors and unleash the sensation of acute pain. Acute pain activates the central nervous system to induce an immediate response to noxious stimulation in the form of a functional protective reaction (i.e. withdrawal, activity avoidance) to protect the injured area and prevent further tissue damage. The resolution of inflammation and recovery of normal tissue function usually results in the cessation of acute pain.

A comprehensive history and medical examination are required to identify a musculoskeletal injury or sinister pathology in the low back pain patient.[24] There are a number of common conditions that are thought to contribute to the development of low back pain. Lumbar intervertebral disc herniation[25,26], spinal stenosis[27,28], lumbar intervertebral joint segmental instability[29], facet joint dysfunction[30,31], and low back muscle strain[32,33] have been theorized to contribute to low back pain. However, in the vast majority of cases seeking medical care, low back pain is considered idiopathic.

Chronic Low Back Pain

Chronic pain has been defined by John Bonica (1990) as "pain that persists a month beyond the usual course of an acute disease or reasonable time for an injury to heal or that is associated with a chronic pathologic process that causes continuous pain or the pain recurs at intervals for months or years.".[34] Chronic low back pain is further described as "nonmalignant pain that has persisted for 6 months or longer despite repeated treatment attempts, is usually experienced daily, and is accompanied by disruption of one or more aspects of the individual's daily functioning.".[35] Chronic low back pain contributes to disability in about 1% of Americans and 3-7% of Europeans.[36] The prevalence of ongoing low back pain tends to be similar for men and women, ranging from 15-30%.[36] Approximately 10% of acute low back pain sufferers do not recover in 3 months and less than 50% will return to work after being off work for 6 months due to persistent pain complaints.[37]

The Development of Chronic Low Back Pain – Risk Factors

Identifying the acute low back pain sufferer who is at risk for developing chronic low back pain is extremely difficult. A number of psychosocial risk factors have been proposed including: fear of pain, pain avoidance, greater anxiety, greater depression, occupational issues (job dissatisfaction, boredom, monotonous jobs, full time work), and maladaptive pain cognitions.[38,39,40] Bigos et al (1991) highlighted the significance of psychological risk factors (job satisfaction, coworker support, psychological job demands, job strain) in work related low back pain complaints.[41] Klenerman et al (1995) collected psychological and physiological data from a sample of 300 acute low back pain patients at three intervals.[42] They reported that the best predictor of the course of low back pain in the first 2 months was the fear-avoidance model, which incorporates stressful life events, personality, previous pain history, and

pain coping strategies. In the multiple regression analyses, the fear-avoidance variables were most successful in predicting pain and disability at two and 12 months.

Persons diagnosed with chronic low back pain tend to demonstrate persistent complaints of pain, pain behaviors (fear of movement, activity avoidance, protective posturing), frequent healthcare utilization, over reliance on pain medication, vocational and financial difficulties, functional impairment, marital and family disruption, and psychological dysfunction with emotional distress.[43] Persons who engage in passive coping strategies and assume the 'sick role' while experiencing a low back pain episode will likely show decreased levels of physical and social functioning with depressive moods and have catastrophizing thoughts. Passive coping behaviors are thought to contribute to activity avoidance and disruption of the daily routine, leading to further physical and psychological dysfunction. This can fortify the persistent pain experience.[44] A recent prospective study by Gatchel (1995) reinforced the importance of psychosocial factors in idiopathic chronic low back pain by showing that physical pathology is not a significant predictor of chronicity in persons with low back pain.[45] A prospective study involving 2487 low back pain patients identified five major risk factors for the development of chronic low back pain including: acute exacerbation of chronic low back pain, sciatica, difficulty walking a short distance or climbing stairs, difficulty rising from bed to chair, duration of sick leave longer than 8 days, and participation in leisure sports.[46] In this study 6.2% of acute low back pain subjects showed a chronic outcome at the seventh week of follow up. Many of the patients with a chronic outcome showed more restriction in their activities of daily living at referral than the subjects who avoided chronicity.

A limited number of physical factors have been found to play a role in the persistence of low back pain. A study by O'Sullivan, Twomey, et al (1998) investigated the abdominal muscle recruitment patterns of chronic low back pain sufferers.[47] Subjects with a history of chronic low back pain showed a functional decrease in the activation of deep abdominal muscles. They suggested this may affect the dynamic stability of the lumbar spine. A comprehensive study of the physical and psychosocial components of chronic low back pain revealed that persons with chronic low back pain showed less trunk flexibility, trunk muscle strength, walking endurance, low back control, and poorer body mechanics during functional testing compared to persons without pain.[48]

It is not known if physical factors (strength, flexibility, endurance, posture) contribute to or result from chronic low back pain. A comprehensive prospective survey of Boeing employees found that strength, flexibility, and aerobic capacity were not predictive of later disability.[49] Activity avoidance and fear of movement have serious physical and psychological consequences for the chronic low back pain sufferer. Protective pain behaviors, in the absence of serious pathology, can perpetuate and prolong the physical and psychological effects of chronic low back pain, contributing to further disability.[50]

Underlying Pathoanatomic Considerations in Chronic Low Back Pain

The pathophysiology of chronic low back pain tends to be elusive. A number of pathologic conditions may persist through a period of acuity and continue to elicit an ongoing pain experience beyond six months. Stenosis, intervertebral disc herniation or degeneration, rheumatologic conditions, chronic lumbar facet joint syndrome, segmental instability or spondylolithesis may be a source of ongoing pain in chronic low back pain sufferers. However, in the great majority of chronic low back pain sufferers the source of the pain is idiopathic.

A meaningful assessment of the low back pain sufferer requires a comprehensive approach, integrating physiological and psychological data. A multidimensional model has been suggested as a framework for low back pain assessment and includes physical ability measures, functional measures, psychological/psychosocial functioning, and pain perception measures.[51]

The Physical Examination of a Low Back Pain Patient

The comprehensive physical therapy evaluation of a person suffering with low back pain typically involves a subjective history and a physical examination. The physical examination plays a critical role in the assessment process. A systematic and detailed physical examination may include an assessment of lumbar spine passive, active, and functional mobility, posture and alignment, trunk muscle function, peripheral joint mobility, neurological testing, intervertebral joint mobility, adverse neural tension tests, sacroiliac joint function, and palpation of soft and bony tissues.[52]

Palpation is applied to identify areas of tenderness, altered temperature, skeletal deformities, and muscle spasm.[53] A study by Waddell et al (1992) investigated the discriminative validity of 27 clinical examination techniques in 120 chronic low back pain sufferers.[54] Posture, spinal tenderness, spinal movement, strength, and pain reproduction were compared between chronic low back pain sufferers and pain free volunteers. The researchers reported that spinal tenderness, straight leg raise, and strength tests were appropriate tests to discriminate chronic low back pain sufferers from pain free subjects.

Despite the widespread use of palpation as an assessment technique, the reproducibility of manual palpation is weak and the quantification of tenderness is difficult to objectify.[55] Katz (1993) reported, "Palpation of the back is generally unrewarding because the structures of interest, including vertebral bodies, discs, nerve roots, and facet joints are buried several inches below the skin.".[56] Cyriax (1987) recommends the use of manual palpation in the low back region to detect spinal irregularities.[57] He suggests that palpation in the low back region that provokes pain in the lower limb may have prognostic value in identifying a neurological lesion that may later require surgical intervention. The assessment of tissue irritability with palpation is not considered to be clinically meaningful unless it is supported with other positive examination data. Magee (1992) suggests that palpation should not be done until the

articular structures in the region of interest have been fully assessed.[22] A study of pain sensitivity by Clauw et al (1999) measured clinical pain (tenderness) at 18 different sites and determined the functional status of 90 chronic low back pain sufferers.[58] The authors argued that an association may exist between tenderness and physical function but did not provide a hypothesis to explain their results. Waddell et al (1982) observed that a reliable examination of tenderness with manual palpation can best be achieved through a systematic and standardized technique.[59] They describe a standardized method of palpating the low back region in the following manner: the patient is relaxed and in prone, the examiner applies slow gentle pressure without causing undue pain, firm pressure is then applied with the ball of the thumb over the spinous processes and interspinous regions, and the examiner asks patient during examination "is that painful?".

In 1980, Waddell et al published an article on "Nonorganic Physical Signs in Low Back Pain" which described a number of physical signs seen in low back pain sufferers that may alert the clinician towards the need for a more detailed evaluation.[60] One of the five nonorganic physical signs investigated was the evaluation of superficial and nonanatomic deep tenderness through manual palpation. Superficial tenderness was evaluated by applying a light pinch over a wide area of the lumbar spine. Deep tenderness was determined by applying a deep pressure over a wide region, extending to the pelvic and thoracic regions. The authors concluded that regional nonanatomic and superficial tenderness, when considered with four other nonorganic signs, "appear to be completely independent of the conventional symptoms and signs of pathologic conditions of the spine.".[60] Nonorganic physical signs were suggested as a tool for identifying those patients that might have a poor surgical outcome.

Since the development of Waddell's nonorganic signs, the relationship between spinal pathology and manual tenderness has been questioned. Recent evidence from pain science research has identified a number of interesting insights into the relationship between tenderness and pathology. Prior to the gate control theory, pain was seen to be either organic or psychological. Physically unexplained pain was considered to have a psychogenic origin, suggesting the pain sufferer's disability lacked a true physiological origin.[61] This dualistic approach to understanding pain persists today despite opposing data derived from the fields of neurobiology and psychology.[62] The nervous system sensitization theory and the biopsychosocial model of pain may help researchers and clinicians to better interpret local and global hypersensitivity demonstrated by chronic low back pain sufferers during the manual palpation portion of the physical examination.

The Anatomy and Physiology of Pain

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".[63] The word pain is derived from the Latin word "poena" meaning penalty or punishment suggesting that pain is purely a

physiological response to noxious stimulation.[64] It is now well accepted that pain is a nociceptive and subjective experience. Pain intensity is most accurately interpreted as the end product of stimulus intensity and personal interpretation based on present and past pain experiences.

Pain begins at the level of the nociceptor. Injured or damaged tissue stimulates mechanothermal and C-polymodal nociceptors with chemical, mechanical, and/or thermal input. Impulses are transmitted along high threshold primary afferent C and Aδ fibers toward the spinal cord. Aδ – fibers are small myelinated fibers that transmit impulses rapidly and are associated with fast, sharp, stabbing pain. C-fibers are small unmyleinated fibers that produce slow, burning or throbbing pain. The cell bodies of C and Aδ fibers are located in the dorsal root ganglion or the spinal cord at laminae I, II, and V. Their axons synapse with second order neurons (wide dynamic range or nociceptive specific neurons) and relay information to supraspinal centers such as the thalamus, brainstem, and cerebellum.[65] The two main ascending neural pathways that transmit pain signals from the periphery to the central nervous system are the spinothalamic and spinoreticular tracts. Other ascending spinocerebral tracts include the dorsal column, spinomesencephalic, and spinohypothalamus pathways. Once pain information reaches the thalamus, pain impulses are widely distributed throughout the cortex (sensory, motor, parietal, frontal, cingulate, occipital) allowing the individual to interpret and understand pain sensation in relation to memories, attitudes, emotions, and beliefs.[66]

In a normal pain-free state, the nociceptive system requires strong stimulation to elicit a pain response. When injury or tissue damage generates sufficient thermal, chemical, or mechanical stimulation to nociceptive activity, tissues at the site of injury may become hypersensitive. Hypersensitivity may also exist in tissues beyond the local area of injury and outside the receptive field of the active nociceptors. Initial and ongoing hypersensitivity may involve the interaction of peripheral sensitization, central sensitization, and biopsychosocial factors.

Peripheral Sensitization

Tissue damage stimulates the release of numerous inflammatory mediators including excitatory amino acids (glutamate, aspartate), neuropeptides (bradykinins, substance P, neurokinin A, calcitonin gene related peptide), and prostanoids (prostaglandins, leucotrienes, hydroxy-acids) from neurons and immune cells.[67] This potent mix of chemicals activates and excites nociceptors in the area of injury.[68] Bradykinins and prostaglandins interact with serotonin, histamine, and cytokines to excite high threshold sensory Aδ and C fibers and propagate the activation of free nerve endings.[67] This "inflammatory soup" resulting from tissue damage sensitizes the primary afferent system and perpetuates local sensory excitation and nociceptor hypersensitivity.[69,70] Increased nociceptor sensitivity is expressed by spontaneous firing, enlargement of the neuronal receptive field, increased sensitivity to mechanical stimulation, and a lowered firing threshold.[71] The maintained availability of chemical mediators and the development of long lasting changes in the sensitivity of the

nociceptor reinforce persistent neuronal excitation.[68] This is termed *peripheral sensitization.*[72] The pain response to mechanical stimulation in this region is enhanced by a decrease in the nociceptive firing threshold and the activation of previously dormant nociceptors.[73] The activation of silent nociceptors contributes to peripheral sensitization by increasing the amount of neuronal discharge from an area of injury in response to stimulation.[74]

Peripheral sensitization may contribute to an upregulated pain state called primary hyperalgesia where the nociceptors of the primary afferent system, in the area of tissue injury, become hypersensitive.[75] Primary hyperalgesia serves a protective function by encouraging the pain sufferer to rest the injured area and allow damaged tissue to heal. Campbell, Khan, and Meyer (1998) examined nociceptor sensitivity following acute injury to test the concept of primary hyperalgesia.[76] Their findings supported the existence of primary hyperalgesia by showing that changes to nociceptor transduction sensitivity occurred only in the area of tissue injury. Kim et al (1995) matched episodic tension type headache sufferers with healthy pain free control subjects and measured pressure pain threshold levels in the head and neck regions.[77] The results from this study suggested that the pericranial muscles of the head and neck showed a decreased pressure pain threshold in episodic tension type headache group compared to pain free controls. The presence of localized pericranial disruption of muscular nociception was supported. Kessler et al (1992) exposed normal *in vitro* mammalian skin-nerve preparation to solution containing inflammatory chemicals (bradykinin, histamine, serotonin, substance P, prostaglandin) and measured the response of nociceptors.[78] They reported that nociceptors were sensitized when exposed to combinations of these substances and the sensitization of primary afferents could be markedly enhanced when preparations were bathed in acidic solutions.

Unfortunately, peripheral sensitization and primary hyperalgesia cannot entirely account for alterations in global pain sensitivity demonstrated in persons suffering from persistent pain. Ongoing pain sufferers have demonstrated secondary hyperalgesia, or hypersensitivity beyond the area of tissue damage, without changes in local nociceptor hypersensitivity.[79] Local nociceptive excitation and peripheral sensitization may coexist with a state of central sensitization resulting in a diffuse, global pain experience.

Central Sensitization

Peripheral sensitization of the primary afferent system bombards neurons in the dorsal horn with afferent impulses. This bombardment activates the release of magnesium blocks from N-methyl-D-aspartate (NMDA) ion channels, allowing glutamate to bind with NMDA receptors and generate a large inward current.[80] Repeated stimulation of NMDA receptors in the dorsal horn allows calcium inflow, which increases nociceptive signal transmission to the central nervous system (CNS) and may overwhelm pain control centers.[81] This alteration in NMDA receptor sensitivity has a profound effect on the intensity, duration, and perception of noxious stimulation.[82] Neuronal plasticity in the dorsal horn and supraspinal regions results

from this neuronal barrage and contributes to spontaneous pain and the expansion of receptive fields. Previously subthreshold inputs from the somatosensory system now begin to generate action potentials from the dorsal horn to the CNS.[83] The term *central sensitization* represents this modulation in central pain pathways contributing to diffuse hypersensitivity in regions beyond the damaged tissue.[84] Central sensitization is reinforced by the activation of low threshold small diameter primary afferents (Aβ – fibers), capable of allowing low intensity stimulation to be perceived as pain and producing long-term changes in spinal neurons.[75]

Ongoing peripheral input is not necessarily required to reinforce persistent pain in a state of central sensitization.[85] Enhanced pain transmission may outlast the initiating noxious input and only low-level peripheral input is required to maintain a painful state.[86] Woolf and Salter (2000) suggest that "Pain is not a passive consequence of the transfer of a defined peripheral input to a pain center in the cortex, but it is an active process generated partly in the periphery and partly within the CNS by multiple plastic changes that together determine the gain in the system.".[86] Flor et al (1997) used magnetic source imaging to investigate the ability of the primary somatosensory cortex to reorganize in chronic low back pain sufferers compared to healthy pain free control subjects.[87] Researchers hypothesized that, "ongoing painful stimulation might result in cortical reorganization due to an excessive nociceptive barrage entering the nervous system.". Painful stimulation was applied to each participant and the early-evoked magnetic field power was measured. Their results demonstrated a shift in the maximum activity elicited in the primary somatosensory cortex from lateral to medial and an enlarged cortical representation in the chronic low back pain subject group. The investigators suggested that, in accordance with previous studies, strong evidence was provided for enhanced cortical reactivity in chronic low back pain subjects. They proposed that somatosensory reorganization may contribute to persistent pain in chronic low back pain sufferers.

In the event of prolonged hyperexcitability and hyperalgesia, a pathological state involving excitotoxicity, cellular dysfunction, and loss of descending control mechanisms may develop.[88] Woolf and Doubell proposed three basic pathological mechanisms that could contribute to central sensitization: increased excitability, decreased inhibition (disinhibition) and structural reorganization of the CNS.[89] Hyperexcitability without adequate descending inhibitory controls may amplify and prolong the pain experience.[90] The combination of excitation and disinhibition might allow pain to persist long after an injury has apparently healed, possible for months or years.[91]

Pain resulting from central sensitization does not serve a protective function and it is not necessarily related to the initial acute injury. Pathological changes in the nervous system may allow pain to persist long after the local tissue injury has healed. The clinical effects of altered pain processing may include false positive physical tests, ectopic impulses, non-dermatomal spread of pain, and unpredictable pain.[92] This clinical data may lead clinicians and patients to determine that previously injured tissues are unhealed. Often patients will avoid activities that might exacerbate their

pain experience and clinicians may continue to search for damaged tissue with hopes of finding an organic source to the patient's persistent pain. Ongoing pain may be related to central sensitization and pathological processing in the CNS rather than a peripheral organic tissue lesion. The exact initiation, maintenance, and control of central sensitization is still under debate but the evidence suggests that central mechanisms interact to strongly reinforce the pathological changes required for persistent pain.

Recent human studies have supported the involvement of central sensitization and altered pressure pain threshold in association with persistent pain conditions. Persons with Juvenile Chronic Arthritis demonstrate extensive global increases in pain sensitivity to noxious stimuli and a decreased pressure pain threshold in affected and unaffected regions compared to healthy pain free control subjects.[93] Complex Regional Pain Syndrome (CRPS type I or type II) sufferers demonstrated a unique pain sensitivity profile when tested with pressure algometry.[94] When pressure pain threshold was compared between CRPS sufferers and pain free volunteers, the CRPS group showed a global increase in sensitivity with lesser tolerance to blunt mechanical pressure. When assessed for pressure pain threshold, fibromyalgia patients demonstrated a significantly higher level of generalized pain sensitivity compared to pain free controls and patients with CRPS.[95] In a study involving chronic whiplash patients, researchers found muscular hyperalgesia in the neck and shoulder region with pressure threshold testing.[14] Increased sensitivity to pressure was also recorded in sites distant from the original area of injury where the subject did not normally experience pain. In a recent study by Dhondt et al (1999) researchers applied pressure algometry to investigate the pain threshold of patients with Rheumatoid Arthritis(RA)[96] Measurements taken bilaterally, at the spine (cervical and lumbar), knee, and ankle demonstrated a significantly lower pressure pain threshold in RA patients at all test points compared to pain free volunteers. The authors suggest that global reductions of pressure pain threshold in RA sufferers may support the contribution of central sensitization to pain in chronic arthritis.

A Biopsychosocial Approach to Chronic Pain

The importance of emotional and psychological factors in persistent pain and disease has been recognized philosophically since the nineteenth century. Hippocrates and Aristotle emphasized the social role of physicians in helping patients cope with illness, pain, and suffering.[97] The obvious inability of a purely physiological model to explain persistent pain highlights the need for a multifaceted approach to pain management. Waddell (1987) applied a biopsychosocial model to explain the difficulties experienced by chronic low back pain sufferers.[98] The biopsychosocial model is based conceptually on the gate control theory of pain, recognizing the importance of individuality and the impact of psychosocial factors on suffering with low back pain.[99] Waddell(1987) suggests that, "pain can no longer be regarded as merely a physical sensation of noxious stimulus and disease, but the conscious experience of pain may be modulated by mental, emotional, and sensory mechanisms and include both sensory and emotional components."[98]

There is a growing body of evidence to suggest that psychosocial and emotional factors play a significant role in the intensity and consistency of pain experienced by chronic low back pain patients. Flor and Birbaumer (1988) demonstrated that chronic pain patients will overemphasize pain by reporting high levels of physical symptoms and are less able to discriminate muscle tension levels in a stressful laboratory situation compared to pain free control subjects.[100] Flor and Turk (1988) examined the relationship between pain related thoughts, controllability, pain severity, and disability levels in chronic low back pain patients.[101] Their results suggested that uncontrollability and helplessness were more closely related to pain and disability levels than disease related variables. Flor, Turk, and Birbaumer (1985) investigated stress related psychophysiological reactions in chronic low back pain subjects.[102] Electromyographic reactivity of the paraspinal muscles, heart rate, and skin resistance were measured in response to personally relevant and general stress in chronic low back pain subjects and compared to healthy pain free controls. Chronic low back pain sufferers demonstrated elevated paraspinal reactivity when discussing personally relevant stress/pain experiences. This study shows the impact of psychological and emotional factors on physiological variables in chronic low back pain subjects. An innovative study by Van den Hout and colleagues (2001) studied the influence of non-pain related failure experiences and pain related fear on pain report in 77 chronic low back pain subject.[103] Researchers determined that pain related fear predicted higher pain reports and resulted in lower pain tolerance in chronic low back pain subjects. Their data reinforced the importance of psychosocial factors in the pain experience.

The influence of psychosocial factors on pain threshold has also been tested in pain free subjects. Anderson and Pennebaker (1980) investigated the importance of stimulus interpretation in pain free volunteers.[104] Subjects were given information about the nature of a pending painful or pain relieving stimulus and then exposed to an affectively neutral vibrating sensation. Participants were then asked to rate the stimulus as painful or pleasant. Researchers found that subjects interpreted the vibrating stimulus as noxious or pleasant in relation to the information provided to them about the stimulus prior to testing. The authors suggested that patients may interpret non-painful stimuli as noxious depending on their experience. A more recent investigation by Rhudy and Meagher (2000) examined the effects of fear and anxiety on pain thresholds in pain free volunteers.[105] Participants were exposed to fear induced by electric shock and to the anticipation of shock, without exposure. Anticipation of shock was designed to induce anxiety. The authors reported divergent effects of fear and anxiety on pain threshold in pain free subjects. Fear was correlated with increased pain thresholds while anxiety was related to decreased pain threshold.

The reported success of various multidisciplinary biopsychosocial treatment approaches in chronic pain management may point towards the influence of biopsychosocial factors in the experience of chronic pain. Guzm, Esmail et al (2001) conducted a systematic review of randomized control trials on patients receiving multidisciplinary treatment for chronic low back pain.[106] They identified 10 studies

that fulfilled all inclusion criteria for a total of 1964 low back pain subjects. All participants received a combination of physical, psychological, and social/occupational rehabilitation for between 30 to 100 hours per week. The systematic review identified that, "there is strong evidence that intensive multidisciplinary biopsychosocial rehabilitation with functional restoration improves function..." and "there is moderate evidence that intensive multidisciplinary biopsychosocial rehabilitation with functional restoration reduces pain...".[106] The ability of cognitive coping strategies to decrease pain reports and improve pain control has also been demonstrated. A detailed meta-analysis of 47 studies conducted by Fernandez and Turk (1988) revealed that 85% of the studies reviewed demonstrated that cognitive coping strategies had a positive effect on pain threshold or pain scores compared to no treatment.[107] Considering the multidimensional quality of chronic pain, an integrated multidimensional approach to treatment should take into account the physical diagnosis, primary and secondary psychological issues, and socially maladaptive pain behaviors.[108] The successes of integrated rehabilitation programs underline the complex nature of chronic pain.

Turk (1996) argued that psychosocial factors might interact with biological mechanisms in two powerful ways.[109] Biological factors may initiate and maintain physical symptoms while psychological issues may influence pain perception and appraisal. Patients then react with pain behaviors in response to their perception of pain. Pain behaviors often include activity avoidance, which eventually leads to physical deconditioning and reduced functional output. As well, psychological elements and cognitive interpretations may alter hormone production, arouse the sympathetic nervous system and increase levels of muscle tension.

The potent influence of biopsychosocial factors on pain threshold has been demonstrated in chronic pain sufferers and healthy pain free subjects. The dominant conceptual approach to chronic low back pain has changed over the years from a medically based model, focused on physical pathology, to a biopsychosocial approach. Global changes in pain threshold and hypersensitivity demonstrated in chronic low back pain subjects might be a by-product of the interplay between emotional, psychosocial, and biological aspects of each subject.[110]

Pressure Pain Detection Threshold (PPDT)

Pressure pain detection threshold has been defined by the International Association for the Study of Pain (IASP) as the lowest stimulus which gives rise to the earliest perception of pain in an instructed subject under given conditions of noxious stimulation.[111] In contrast, pain tolerance is defined as the highest level of noxious stimulation tolerated by a subject.[112] Due to obvious ethical and practical implications, pain tolerance is rarely tested in experimental pain sensitivity studies.

Pain threshold measurements attempt to quantify sensory discriminability and response bias related to a subject's pain report. Discriminability measures the ability of a subject to differentiate variations in stimulus intensity. Response bias attempts to assess the willingness of a subject to report pain following stimulation.[113] To clearly

understand a subject's pain processing researchers focus on controlling response bias and measuring the subject's discriminability. Alterations in pressure pain threshold or pain discriminability may signal changes in the ability of the subject to process painful stimuli.[112]

Typically, in a state of good health, without background pain, the intensity of pain experienced is directly related to the intensity of stimulation and the localization of pain directly reflects the site of stimulation. Population studies have shown that pain threshold levels may vary between individuals, but tends to remain constant within each subject.[114,58] Pain threshold represents a continuum with some individuals showing a high threshold and others a low threshold with most pain threshold scores clustered around the mean. The mean pain threshold represents a state of normosensitivity of pain. Alterations in this normosensitive pain state can lead to either hypersensitive or hyposensitive pain states.[115]

Beecher (1959) suggested that the pressure pain threshold in healthy subjects can be influenced by a number of confounding factors including: race, gender, age, anxiety, subject/observer interactions, distraction, dominant hemisphere, past experience, and personality.[116] A study by Rhudy and Meagher (2000) examined the effects of fear and anxiety on pain reactivity in human subjects by measuring changes in pain threshold following exposure to anxiety and fear.[105] The researchers found: (1) Subjects who are afraid of threatening events tend to show an increased pain threshold; (2) Subjects that are anxious in anticipation of a threatening event will experience a decreased pain threshold. Ohrbach and colleagues (1998) investigated the effect of examiner expectations on the measurement of pressure pain detection threshold.[117] The authors concluded that examiner expectancy might affect the magnitude of pressure pain threshold but not the reliability.

Gender Influences on Pain Threshold

The divergent effects of gender on pain threshold have been studied extensively in pain science. Keogh and Birkby (1999) explored the effect of anxiety and gender on the experience of pain in 93 pain free participants (55 females and 38 males).[118] In this study, pain was experimentally induced with a cold pressor task and the McGill Pain Questionnaire was utilized to measure the pain report of each subject. Their results demonstrated a lower pain threshold and lower tolerance to cold pressor pain in females compared to males. The researchers suggested that males might be reluctant to admit pain even though their pain experience might be similar to female subjects. Fillingim et al (1999) explored the experience of clinical and experimental pain among female and male subjects.[119] In this study, two hundred and nine (117 females and 92 males) healthy pain free adults underwent testing to determine and compare their thermal pain threshold and thermal pain tolerance levels. Thermal pain testing revealed a greater sensitivity to thermal pain in females compared to males by demonstrating a significantly lower pain threshold and pain tolerance levels in females. Female subjects reported greater healthcare utilization and more pain sites over the preceding months yet no difference was reported in the number of pain

episodes between genders. Numerous other studies have demonstrated consistently lower pain threshold levels in female compared to male subjects in response to clinical and experimental pain.[120,121,122,123 124,125,126]

A major confounding factor in determining difference in pain threshold between male and female subjects is the gender of the experimenter. Levine and De Simone (1991) studied the effects of experimenter gender on pain report in 68 pain free subjects (35 male and 33 female).[127] A cold pressor test was applied and subjects were asked to relate their pain experience on a Numeric Rating Scale (1-32) and the Pain Affective Scale using affective words. Subjects were randomly assigned to a male or female experimenter. Experimenters were undergraduate students, blinded to the hypothesis, and selected to accentuate the stereotypical gender characteristics of masculinity and femininity. The results of the experiment showed that male subjects reported lower pain scores (higher pain threshold) when tested by a female observer versus a male observer. Female subjects demonstrated consistent pain intensity scores when tested by female and male observers. No experimenter effects were found when male or female experimenters tested female subjects.

Pharmacological Influences on Pain Threshold

The effect of opiate analgesic medications on pain threshold has been repeatedly tested. Generally, opiates such as Morphine, Demerol, and Codeine have been found to increase pain threshold thereby decreasing sensitivity. Enggarrd et al (2001) tested the analgesic effect of codeine compared to imipramine by applying pressure, electrical, and cold stimulation to 18 pain free volunteers.[128] Medicated participants were compared to placebo-controlled subjects for differences in pain threshold. Codeine significantly increased the pressure, electrical, and cold pressor pain threshold levels of subjects. Imipriamine increased the pressure pain threshold slightly but did not have a significant effect on the electrical pain threshold or tolerance to cold pressor tests. Jones et al (1988) used a cold pressor test to compare the pain relieving effects of morphine to ibuprofen in 12 pain free subjects.[129] The researchers observed a significant pain intensity decrease and pain tolerance increase in subjects who received morphine. Participants receiving ibuprofen showed similar changes in pain intensity and threshold to individuals who received placebo medication. Opiate derived medication demonstrated a more powerful effect on pain tolerance and pain intensity ratings than the non-steroidal, anti-inflammatory drug ibuprofen. The results of these investigations have been confirmed by other studies that have demonstrated the ability of opioid analgesics to decrease pain intensity rating and increase pain tolerance.[130] Limited research has been dedicated to determining the effect of withdrawal or narcotic addiction on pain threshold. Tilson, Rech, and Stolman (1973) assessed the effect of cessation on the pain sensitivity of morphine users.[131] They reported that increased pain sensitivity and hyperexcitiability may occur following the abrupt cessation of morphine in opioid dependant subjects. The authors suggest further that the intensity of withdrawal symptoms may be related to the degree of physical dependence.

Physicians frequently prescribe tricyclic anti-depressant medication to help patients manage chronic pain. Fishbain (2000) conducted a systematic literature review to determine the pain relieving effects of anti-depressant medication in chronic low back pain subjects.[132] The systematic review demonstrated that 7 of 10 trials administering sertonergic-noradrenergic antidepressants, to chronic low back pain subjects, reported a decrease in pain report. As well, four of five trials administering noradrenergic anti-depressants, to chronic low back pain subjects, showed a decrease in pain intensity. Generally, anti-depressants were found to reduce chronic low back pain reports in the majority of subjects. Bendtsen and Jenson (2000) investigated the effects of amitriptyline on myofascial tenderness in 33 subjects with chronic tension headache.[133] Pressure pain detection threshold and tolerance was recorded at the finger and the temporal regions. Amitriptyline reduced tenderness and headache intensity significantly more than placebo but it did not affect pressure pain threshold at either of the examined sites. Researchers suggested that amitriptyline elicits its analgesic effect in chronic myofascial pain by reducing the transmission of painful stimuli from myofascial tissues rather than by reducing overall pain sensitivity of the subject. Poulsen et al (1995) studied the hypoanalgesic effect of imipramine on thermal and pressure pain thresholds, withdrawal to electrical stimulation, and continuous pain rating to a cold pressor test in 12 healthy pain free volunteers.[134] Imipramine did not affect heat or pressure pain detection threshold measurements, but it did affect thermal and pressure tolerance levels. Pain ratings to the cold pressor test were unaltered by imipramine. The administration of tricyclic anti-depressant medication has been shown to preferentially affect pain reports, but it has not been consistently shown to effect pressure pain detection threshold in persons with chronic pain.[133,135,136]

The literature is unclear on the effects of anticonvulsant medication on pain threshold levels in human subjects. Yang et al (1979) explored the analgesic actions of diazepam in 20 young healthy volunteers.[137] They reported a much less significant effect on pain threshold compared to morphine. Wiffen et al (2001) designed a comprehensive and systematic literature review to study the effects of anticonvulsant medication on acute and chronic pain.[138] The results of this review showed that anticonvulsant medication does not provide effective analgesia for persons with acute pain. Anticonvulsants have been widely recommended and administered for the management of neuropathic pain (i.e. diabetic neuropathy, trigeminal neuralgia, post-herpetic neuralgia) and have proven to be effective in managing neuropathic pain conditions.[139,140]

Non-steroidal anti-inflammatory (NSAID) medication is used widely by persons with low back pain for analgesia and inflammatory control. A recent review by van Tulder et al (2001) assessed the analgesic effects of NSAIDs in persons with non-specific low back pain.[141] Non-steroidal anti-inflammatory medication was compared to muscle relaxants, narcotics, acetaminophen, Vitamin B, non-drug treatments, and other NSAIDs on the basis of pain relief. The reviewers concluded that, "...NSAIDs are effective for short term symptomatic relief in patients with acute low back pain...Sufficient evidence on chronic low back pain is still lacking.". The

effect of NSAIDs on pain threshold was not discussed. Walker and Carmody (1998) investigated the effects of gender and ibuprofen on pain threshold in 20 healthy age matched pain free subjects.[142] The researchers reported that neither ibuprofen nor placebo had any effect on pain threshold. In contrast, Dragani et al (1988) investigated the effects of oral Diflunisal on the electrical pain threshold of 12 pain free subjects.[143] Their results showed that pain threshold was significantly increased two and three hours after 500mg of diflunisal and one, two, and three hours after 1 gm of diflunisal. The authors concluded that diflunisal has the ability to increase pain threshold in healthy human subjects.

Medication exerts a powerful influence over pain intensity reports in persons with acute and chronic pain. Narcotics have been shown to increase pain threshold and decrease pain complaints in persons experiencing pain. Non-steroidal anti-inflammatory and tricyclic anti-depressants drugs have been shown to decrease pain reports and may have an effect on pain threshold. Anticonvulsant medication does not affect acute pain complaints but may exert an analgesic effect in persons reporting chronic pain. The effect of NSAIDs, anti-depressants, and anticonvulsants on pain threshold has not been fully addressed in the literature.

Worker's Compensation Insurance and Pain Threshold

The impact of Worker's Compensation Insurance involvement on disability and pain has been investigated extensively over the past few years. It has been suggested that individuals receiving compensation payments report higher pain perception levels than similar persons in pain and not receiving compensation. A meta-analysis of 32 studies investigated the overall impact of compensation on the nature and intensity of pain reported by subjects with chronic pain.[144] The authors reported that the literature clearly demonstrates persons receiving compensation payments report a heightened overall pain experience and a reduction in the ability to benefit from medical or psychological treatment compared to subjects not receiving compensation. A more recent study by Turk and Okifuji (1996) investigated the roles of physical findings, financial compensation, and the mechanisms leading to pain on adaptation and emotional distress in chronic pain subjects.[145] One hundred and fifty-eight subjects referred to a multidisciplinary pain clinic were subjected to a three hour pain evaluation including a Multidimensional Pain Inventory (MPI) to determine pain severity, perceived interference of pain in activities of daily living, affective distress, perceived control over life, support from significant others, responses by significant others, and the performance of a set of common activities. The results of the study reported that, when receiving compensation, chronic pain sufferers exhibited greater psychological distress, higher pain severity, and greater life interference. As well, objective physical signs were not significantly correlated with pain severity. Numerous other studies support the notion that persons with chronic pain, receiving compensation payments, demonstrate greater disability, pain behaviors, and higher levels of perceived pain.[146,147,148]

Pain Threshold Research and Low Back Pain Subjects

Peters, Schmidt, and Van den Hout (1989) measured the pressure pain perception (detection) threshold, pain tolerance, and pain intensity of 20 male chronic low back pain participants and 20 age-matched control subjects.[149] The researchers applied pressure to the middle phalanx of the second finger on the non-dominant hand of each subject with a modified Forgione-Barber pressure stimulator. Pain perception (detection) threshold and the pain tolerance levels were measured over eight trials. Their results revealed a trend towards higher pain thresholds in chronic low back pain subjects but the differences between groups were not statistically significant. The control group tended towards low pain thresholds in the first trial and progressively higher thresholds in subsequent trials, increasing to the 8th trial. The chronic low back pain group did not demonstrate a similar pattern. A statistically significant difference in pain thresholds between chronic low back pain and pain free subjects groups was not found, perhaps in part due to large inter-individual variations in pain threshold and tolerance.

In another study of pain threshold in chronic low back pain sufferers, Peters and Schmidt (1992) compared 20 subjects with chronic low back pain (12 males, 8 females) with 23 healthy pain free control subjects (13 males, 10 females). Subjects were evaluated in terms of three variables: pain threshold, pain tolerance, and pain discrimination. Pain tolerance was measured at the right ankle with an electrical stimulus. Pain threshold was measured by applying pressure to the middle phalanx of the second finger with a Forgione-Barber pressure stimulator. The Forgione-Barber pressure stimulator is a device consisting of a dull Lucite knife edge (.25 mm thick) attached to an adjustable lever arm. The knife edge exerts a constant pressure on the second phalanx of any finger, resulting in a sensation of slight pressure that gradually builds into a dull, aching pain. Changing the magnitude of weight attached to the lever arm can vary the rate at which the sensation becomes painful. Pain discrimination was measured using a forced-choice discrimination task. All subjects received pain tolerance, pain threshold, and pain discrimination testing.

The results of this investigation showed that chronic low back pain sufferers were generally less sensitive to experimental pain (pressure and electrical), both in terms of pain threshold and pain tolerance. The researchers suggested two possible explanations for the decrease in pain sensitivity identified in their data. First, it was suggested that chronic low back pain sufferers may tend to perceive experimental pain as comparably mild in light of their past pain experiences. This suggestion sides with the "adaptation theory" of pain modulation. The second explanation is based on the "pain inhibits pain" principle which argues that two concurrent painful stimuli could form a noxious inhibitory mechanism leading to decreased sensitivity in chronic low back pain sufferers. A significant limitation of this study was that the authors assumed that pain threshold measurements taken at the second finger of chronic low back pain subjects adequately represented each subject's pain perception threshold.

Literature Review Summary

Low back pain has reached epidemic proportions throughout most of the western world. Acute low back pain affects nearly 80% of persons and contributes to billions of dollars in healthcare and work-related expenditures. Most acute low back pain sufferers experience intermittent pain exacerbations that usually resolve spontaneously. A small percentage of acute low back pain sufferers experience ongoing low back pain persisting beyond twelve weeks. Persistent low back pain may have physical, psychological, and social implications and may interfere with all aspects of the sufferer's life. Many individuals complaining of low back pain will seek the advice of a health professional. The physical examination of a person suffering with low back pain is complex and usually involves the evaluation of articular, neurological, and soft tissue structures. Soft tissue palpation is often applied by clinicians to confirm soft tissue irritability and detect superficial skeletal irregularities. A pressure threshold meter has been developed to quantify palpatory tenderness and is typically the tool of choice for determining the pressure pain detection threshold. The pressure pain detection threshold (PPDT) is the least amount of pressure that can be perceived as painful in a given subject. Pressure pain detection threshold tends to be susceptible to subject and examiner bias. When properly controlled, the pressure threshold meter has been found to be a valid and reliable tool for quantifying the pressure pain detection threshold.

The study of neurobiological pain mechanisms has exploded over the past decade and continues to gain momentum in medical and rehabilitation research. Generally, pain can be protective and functional or maladaptive, without a protective purpose. Protective pain will sound an alarm, alerting the central nervous system (CNS) to the presence of tissue damage. The somatosensory system transmits this information to the dorsal horn which relays information to the supraspinal centers. Typically, a response will be coordinated by the CNS to avoid painful activities and protect the injured area. Following soft tissue or nerve injury, a pathological state may develop in which the pain sufferer may demonstrate local hypersensitivity, persistent pain, or the spread of pain to uninjured tissue. Peripheral sensitization represents this alteration in nociceptive sensitivity which tends to involve spontaneous firing, enlargement of the receptive field, and a decrease in pain threshold. Central sensitization may result from a persistent increase in afferent traffic to the CNS and may lead to profound changes in pain transmission. Central sensitization may contribute to prolonged protective pain behaviors having no functional advantage. Persistent pain and hypersensitivity may be reinforced by central mechanisms, through structural reorganization. Ongoing nociceptive input may not be a necessary requirement for persistent pain.

A purely physiological, one-dimensional approach towards understanding global hypersensitivity in persons with chronic pain has failed to provide a consistent and satisfactory explanation for ongoing pain symptoms. The biopsychosocial model suggests that emotional, social, and psychological factors interact with biological factors to create changes in the chronic pain sufferer's pain threshold. Anxiety, stress, learned helplessness, and expectations play a significant role in pain perception and

can have a dramatic impact on sensory experiences. In support of the biopsychosocial model, cognitive and psychosocial treatment strategies have been shown to reduce pain complaints and increase function in persons with chronic pain.

It is most likely that neurobiological and biopsychosocial pain models collaborate, at varying degrees, to produce ongoing fluctuations in the sufferer's pain experience. The dominance and/or inhibition of certain biological, physiological, emotional, and psychosocial factors likely result in the relief or exacerbation of ongoing, persistent pain.

Gender, medication, and compensation status have each been shown to affect pain complaints and/or pain threshold in persons with pain. Female subjects tend to demonstrate a lower pain threshold compared to males. The gender of the experimenter may also influence pain threshold. Medication usage also has an impact on pain intensity reports and pain threshold. Narcotic, anti-inflammatory, anti-depressant, and anti-convulsant medications each have a unique analgesic effect on pain threshold or pain intensity reports. Subjects receiving compensation for disability of injury tend to show greater pain severity, psychological distress, and life interference.

Research Methods

This study compared pressure pain detection thresholds between three groups of subjects with different classifications of low back pain. The advantage of this non-equivalent group design is that intact groups, considered to be similar, were selected and compared for differences on a specific variable. This quasi-experimental design, although simple, is susceptible to threats of external validity (sampling bias) and internal validity (selection bias – assembly, history; measurement bias – subject error, rater error, and errors in instrumentation) that may have distorted or invalidated the results of this study.[150] This research project was designed to avoid or minimize such threats.

A non-probability purposive sampling strategy was applied in this research project. Non-random subject selection is simple and consumes fewer resources (time, money) than random sampling. However, non-probability sampling is susceptible to sampling bias and may lack generalizability. Sampling bias was minimized in this study by selecting low back pain subjects from the offices of a variety of care providers including family physicians, physical therapists, and chiropractors. Subjects were recruited by their healthcare practitioner and then referred to the principal investigator for further discussion and screening. A small number of acute low back pain subjects were recruited through a newspaper advertisement near the completion of the study.

In the design of this project, the primary threats to internal validity were selection bias and instrumentation. The past medical history of each subject may have affected their pressure pain detection threshold and was controlled through stringent

subject screening to ensure all subjects met the inclusion criteria prior to acceptance into the study. Instrumentation can be a threat to internal validity when a measurement tool used for data collection requires calibration after a certain amount of use. The pressure threshold meter employed in this project, supplied by JTech Medical, was calibrated and certified for accuracy by the manufacturer (see Appendix N). The manufacturer asserts that this assessment tool does not require regular re-calibration. Rater error also could have affected the experimental outcome of the study if the examiner added her interpretation or bias towards a desired response. A trained female examiner, blinded to the subject's assigned group and the expected results of the study, was instructed to adhere to a strict testing protocol and to avoid unnecessary interaction with subjects. In a similar way, subject error could have affected the measured response if subjects were biased towards helping or hindering the outcome of the study or if the subject was afraid of the measurement technique. A careful description of the testing procedure was given to each subject and a 'trial run' on the mid-thigh was performed to help reduce the risk of subject error. Preventing subjects from viewing PPDT measurements obtained during testing further controlled subject error. General background information about the study was disclosed to each subject, but the experimental hypotheses of the project or trends in the literature were not discussed.

The presence of confounding factors, potentially affecting the outcome of PPDT, may not have been equally distributed in acute and chronic low back pain sufferers and therefore efforts were made to control these in the study design. Control for confounding factors was accomplished by limiting the study to female subjects, excluding subjects with pre-existing or concurrent pain conditions, monitoring and possibly excluding subjects using certain medications prior to testing, and encouraging subjects to avoid physical treatment within 24 hours of testing. Respecting the ongoing literature debate on the relationship between compensation and pain sensitivity, low back pain group participants were excluded if they were involved in a Worker's Compensation or disability insurance claim. This control was implemented to ensure that the potentially confounding effects of compensation did not alter and bias PPDT measurements. Finally, in the community where the study was conducted, it was the general sense among the medical community that females were less likely to sustain a work-related low back injury. Group evidence for comparability was collected and reported for those variables that were not matched (i.e. age).

Subject Group Characteristics

Three groups of subjects were recruited based on the following inclusion criteria:

1. Acute Low Back Pain Group

    a. Low back pain with or without leg pain extending below the knee, consistently experienced for *less than 3 weeks*,

b. Moderate or greater severity of pain as judged from a numeric pain rating scale (≥4/10),

2. Chronic Low Back Pain Group

a. Low back pain with or without leg pain extending below the knee, consistently experienced for *more than 6 months*,
b. Moderate or greater severity of pain as judged from a numeric pain rating scale (≥4/10),

3. Pain Free Group

a. No low back or leg pain during a 6 month period prior to testing,

Additional inclusion criteria for all three groups were:

a. Female,
b. 20 to 60 years of age,
c. Free of concurrent painful conditions (i.e. neuropathic pain conditions, phantom limb pain, complex regional pain syndrome, spinal cord injury, fibromyalgia, migraine headaches, head injury, malignant disease or other painful conditions), as well as pregnancy and head injury,
d. Have no communication deficits that would interfere with testing (i.e. hearing problems, unable to speak or understand the English language),
e. Have not consumed Opioid Analgesics (i.e. Morphine, Demerol, and Codeine), benzodiazipine, antidepressant, or anticonvulsant medication within the past 3 weeks. Non-steroidal anti-inflammatory (i.e. Naproxen, ASA) and other analgesic (i.e. Ibuprofen, Tylenol, Advil, Acetaminophen) medication must have been discontinued 24 hours prior to testing, but can be resumed immediately following testing,
f. Have not had physical treatment (i.e. physiotherapy, chiropractic, TENS) 24 hours prior to testing,
g. Subjects must be able to safely tolerate prone or side lying positions,
h. Subjects must not be involved in a disability insurance claim (Worker's Compensation or other disability insurance).
i. Have not experienced low back surgery within six months of testing.

The inclusion criteria noted above were imposed to control for several potentially confounding variables including gender, medication use, recent physical treatment, concurrent painful conditions, and involvement in an insurance claim.

Subjects were not asked to discontinue taking pain medications to participate in this study. Rather, subjects were recruited who were not presently taking the specific medications noted above (e). Subjects taking NSAIDs or over the counter analgesics were informed that if they discontinued using medication within 24 hours of testing they may experience an increase in pain until they resumed taking the medication.

The inclusion and exclusion criteria were also based on the protection of subjects and the scientific merit of the investigation. Female subjects were selected for this study for a number of reasons. Sample size calculations suggested that, for adequate power in data analysis, 30 subjects must be included in each of the three groups (Acute, Chronic, Pain Free). The minimal numbers of subjects were used in this study; ensuring adequate scientific rigor and minimal harm to the fewest number of subjects (see Appendix A). The literature unequivocally suggests that the pain threshold of male and female subjects varies significantly. Therefore, male and female subjects had to be divided into separate groups. Recruitment and funding for 180 subjects for this size of project would exhaust both time and economic resources, therefore either male or female subjects had to be chosen. Historically, females have been understudied. As well, in the community where the study took place, females tend to have fewer WCB related low back claims. Finally, the literature has also shown that subject-observer effects are apparent during pain threshold testing.[127] Specifically, when tested by a female examiner, male subjects tend to show a higher pain threshold than when tested by a male. Female subjects do not show significant differences in pain threshold when tested by a male or female observer. The research assistance selected to perform the test procedure in this study was female. Considering the information given above, female subjects were selected for testing in this research project.

Subject Recruitment:

A 'sampling of convenience' strategy was applied to the recruitment of all subjects for this study. Acute and chronic low back pain subjects were recruited from physician offices, physical therapy clinics, and chiropractor clinics in the Lloydminster area. Each physician was given an information package containing all the relevant documents pertaining to the research project and inclusion criteria for subjects with low back pain. In addition, informal discussions were carried out with each individual physician to discuss the nature of the project, and encourage participation by the physician. The primary investigator visited each chiropractic and physical therapy clinic, providing each clinician with an information package with detailed information concerning subject recruitment and a general description of the research project. Inclusion criteria were explicitly discussed, reinforcing the importance of early recognition of patients with acute low back pain. Physicians, physical therapists, and chiropractors received a clinician-screening checklist (see Appendix K) describing the inclusion criteria and a subject handout (see Appendix I) to give to potential subjects. A tear off portion on the bottom of the subject handout was provided for potentially interested subjects to leave their name and number for the primary investigator with the receptionist at each clinic. Potential subjects either contacted the primary

investigator directly or their contact information was forwarded to the primary investigator for subjects to be contacted at a later date. The primary investigator screened potential subjects during the initial phone contact to ensure inclusion criteria were met prior to attending a testing session. The primary investigator discussed the project with local physicians on a regular basis through the data collection phase. The primary investigator also visited physical therapist and chiropractic clinics regularly to encourage participation, express appreciation for their assistance, and answer any questions. Additional information was provided to physicians, physical therapists, and chiropractors on three separate occasions to remind clinicians about the study and reaffirm the inclusion criteria applied to each subject group. Pain free subjects were recruited through a district wide newsletter, posters, and word of mouth (see Appendix O).

The recruitment of subjects suffering with acute low back pain was difficult. Once testing was complete on the 'Chronic Low Back Pain' and 'Pain Free' subjects, only a small number of 'Acute Low Back Pain' subjects had been recruited. Exhaustive efforts were put forth to recruit acute low back pain sufferers. Several advertisements were presented in the local newspaper and an advertisement was publicized in a daily news pamphlet every other day for two months (Appendix P). Once recruitment was complete for the 'Chronic Low Back Pain' and 'Pain Free' groups, clinicians were only provided with information specific to the "Acute Low Back Pain' group. Posters were provided to each clinic to advertise for acute low back pain sufferer volunteers. In addition, posters were also put up in the Emergency Department of the Lloydminster Hospital and throughout the local college. Two subjects with 'Acute Low Back Pain' were recruited with these efforts. In addition, the principal investigator discussed the need for "Acute Low Back Pain' sufferers with physicians, physical therapist, and chiropractic clinicians on a very regular basis.

Various reasons for the difficulty experienced in recruiting "Acute Low Back Pain" sufferers were expressed by the clinicians and noted by the principal investigator. Recent research has demonstrated that care-seeking by acute low back pain sufferers is low. Vingard, et al (2002) reported that in a population of 17,000 men and women, between the ages of 20 and 59, approximately 5% of acute low back pain sufferers sought care during a three year period.[151] Conversely, chronic low back pain sufferers often have an ongoing relationship with one or more healthcare providers in order to receive rehabilitation, medications, specialist referrals, or diagnostic testing. Chronic low back pain sufferers tend to seek care more often than acute low back pain sufferers.[152] Carey et al (1999) reported that care seeking was more common (87%) in persons with severe and recurring low back pain than subjects having mild or no recurrence of low back pain.[153] Acute low back pain sufferers may not seek care since the vast majority of acute low back pain is self-limiting and benign.[154] As well, in the community where this study was conducted, waiting lists to see a physician or physical therapist can range from 1-3 weeks, depending on the severity of the condition. If the acute low back pain sufferer waited on a list for more than three weeks, she no longer qualified to participate in this study. Jacob, Zeev, and Epstein (2003) reported that 28.6% of acute low back pain sufferers turn to alternative medicine (acupuncture,

reflexology) for pain relief rather than traditional medicine.[155] Alternative medicine practitioners did not participate in this investigation. Acute low back pain sufferers may also be more inclined to take pain medication than chronic low back pain sufferers. It may be that persons with acute pain are less able to cope with pain, than chronic pain sufferers, and typically require pain medication to ease the burden of pain sensations. Another possible reason for the difficulty experienced in recruiting acute low back pain sufferers for this study was that the design of the project did not offer direct physical benefit (reduction of symptoms) to the potential subject. Therefore, acute low back pain sufferers may have been reluctant to participate in a study that would not assist in relieving their symptoms or managing their pain condition.

The number of subjects and characteristics of subjects who chose not to participate in the study is not known. Subjects who showed interest in the study, but were excluded according to the inclusion criteria provided in the section above were counted.

All subjects received a modest remuneration of $10 for their participation in the study. Free parking was provided for each subject.

Sample Size

A goal of 90 participants was established with 30 subjects per group (see Appendix A for calculation of sample size). Sample size was based on a α level of .05 and β level of .10.

Instrumentation

The Pressure Threshold Meter – Clinical Considerations, Reliability, and Validity

Pain provocation through manual palpation is a common technique, employed by the clinician and the pain sufferer, to ascertain the location and severity of injury or the success of physical or pharmaceutical treatment. The determination of pressure pain threshold tends to be subjective, and susceptible to examiner bias.[156] Examiners may interpret force magnitudes differently, various sized surfaces may be applied to the subject's skin, and consistent measurement techniques may not be utilized between test sites or repeated test sessions. To minimize examiner bias, a pressure threshold meter (PTM) has been developed.

The pressure threshold meter allows for the quantification of pressure pain detection threshold (PPDT), for scientific and clinical applications. The PTM is simple to apply and can quickly provide accurate information on a patient's level of pain sensitivity or tissue irritability. The PTM measures mechanical pain similar to that encountered with tissue stretching and therefore tends to be more clinically relevant than thermal or electrical pain stimuli. Furthermore, PPDT measurements with the PTM do not rely on the subject's verbal or academic abilities, they avoid cultural bias, do not evoke fear in the subject, and have proven to be a fast and reliable pain measurement tool.[157]

A pressure stimulus is preferable over thermal and electrical stimulation, for the determination of pain threshold in painful and pain free groups for the following reasons: (1) Mechanically evoked pain is more clinically relevant than thermal or electrical pain; (2) The application of pressure is simple and convenient; (3). Mechanical pain is possibly more familiar to subjects and may provoke less fear in the subject during and prior to testing compared to thermal or electrically induced pain.

Reliability of the Pressure Threshold Meter

The reliability of PPDT measurement considers the extent to which pain threshold measurements with the PTM are repeatable, and indicates the amount of confidence anticipated by researchers in PPDT measurements.[158] The pressure threshold meter has been repeatedly tested, in both healthy and painful subjects, and has been deemed a reliable tool for the quantification of pain sensitivity or relative tenderness.[159] A summary of the reliability of the pressure threshold meter is given in Table 1.

Table 1: **Reliability of the Pressure Threshold Meter**

Reference	Intra-observer Correlations	Inter-observer Correlations
Reeves et al (1986)	0.69-0.91	0.76-0.89
Merskey and Spear (1964)	0.59	0.65
Chung et al (1992)	0.67-0.92	0.49-0.77
Hogweg et al (1992)	0.74-0.93	0.56-0.89
Nussbaum and Downes (1998)	0.94-0.98	0.74-0.89 (single) 0.81-0.86 (mult)

Reeves et al (1986) tested the reliability of a PTM in three studies of myofascial trigger point sensitivity.[122] Study one involved PPDT measurements, by two investigators, with a PTM in 15 subjects reporting head and/or neck pain at five trigger points in the head and neck region and one non-myofascial bony point. Pearson correlations were calculated for between and within experimenter reliability. The resulting r-values ranged from $r = 0.692\text{-}0.909$ for within experimenter correlations and $r = 0.761\text{-}0.893$ for between experimenter correlations. All correlations were significant ($p<0.01$), supporting a high between and within experimenter reliability. Study two evaluated the PPDT in 12 patients with myofascial pain of the head and neck region using a PTM. The same two experimenters took only one measurement at each site and the test sites were not marked. A Pearson correlation was computed and all correlations demonstrated reliability ($P<0.05$) except measurements at the bony test site. Correlations ranged from $r = 0.670\text{-}0.860$ for soft tissue test sites. The authors reported that experimenters were able to consistently locate and measure the same unmarked points. Studies three measured PPDT, with a PTM, in nine subjects having head and/or neck pain at five test points and at sites 2 cm adjacent to the myofascial trigger points tested in studies one and two (same sites as

studies one and two without the bony test site).The same two experimenters measured the PPDT only once at each marked trigger point and adjacent non-trigger point. Test results showed a statistically significant decrease in pain sensitivity at sites 2 cm from the trigger point locations ($p<0.05$). The researchers suggested the results of study three might argue for the validity of trigger point sensitivity measurement. In general, this three part study demonstrated the reliability of pressure algometry as a measuring tool for trigger point sensitivity.

Reliability of the pressure algometer was also studied by Merskey and Spear (1964).[125] Forty-nine medical students underwent pressure threshold testing, on the forehead, by 2 observers on 2 different occasions to determine intra-observer and inter-observer reliability of the pressure algometer. Pearson Correlations reported by the authors were $r = +0.65$ between two observations by the same examiner and $r = +0.59$ between examiners. Both correlations were highly significant ($p< 0.01$).

Buchanan and Midgely (1987) applied a simple hand operated PTM to measure pressure thresholds at sites throughout 18 high school students.[123] One examiner tested five sites in each subject with five minutes between each measurement. The researchers reported that a significant difference was not found between the two repeated measurements for each subject ($p<0.05$). A high degree of intra-observer reliability was suggested by the authors, when a single observer performed pain threshold testing. As with previous studies, the researchers agreed that the rate and constancy of pressure application were important aspects of the test procedure although they had no means of calculating these with their measurement technique. Reliability was determined based on intra-observer error between two observations in all five body regions. Correlation or kappa values were not provided by the authors.

Another investigation by Fischer (1987) endeavored to quantify the pressure pain threshold over normal muscles in 50 pain free subjects (24 males and 26 females).[160] He applied a PTM to eight bilateral points on the neck, shoulder, and back. The resulting PPDT measurements were compared between right and left sides within each subject. All differences noted between right and left sides, in both males and females, were non-significant ($p<0.05$) except for the supraspinatus muscle in female participants. Considering the similarity of bilaterally PPDT tests within each subject, the investigators argued that excellent reliability with the PTM was demonstrated. Correlation or kappa values were not provided by the authors.

In a study by Chung et al (1992) researchers applied a new electronic algometer (PTM) to measure PPDT in 40 dental students at 13 well defined points in the facial, masticatory, and neck muscles.[161] Pearson correlation coefficients between and within examiners were ascertained. For female subjects, correlation coefficients ranged from $r = 0.671$-0.921 within examiners and $r = 0.489$-0.768 between examiners. Evidence of high intra-rater and inter-rater reliability was obtained in all head and neck muscles ($p<0.01$) except points in the medial pterygoid and middle sternocleidomastoid muscles. The authors suggested that it was very difficult to hold the algometer perpendicular to the medial ptyergoid muscle. This may account for the

poor correlation coefficients recorded for inter-rater and intra-rater reliability. The electronic algometer was also poorly suited to testing the middle sternocleidomastoid muscle due to its placement over visceral structures. A pincer type algometer was recommended for testing this muscle. Aside from the logistical difficulties experience with testing the pterygoid and sternocleidomastoid muscles, the investigators recommended the use of a electronic pressure algometer for the evaluation of muscle tenderness in clinical and experimental studies.

Hogeweg et al (1992) examined the pressure pain threshold of peripheral joints (upper and lower) and paravertebral tissues bilaterally in 28 pain free individuals (14 male and 14 female).[120] Two observers measured PPDT in all subjects, using a PTM, in two separate rooms in sequential order. Each test site was measured three times and the mean was calculated to represent the PPDT at that specific point. Pearson correlation coefficients were calculated to test inter-observer differences and laterality. The authors reported correlations ranging from $r = 0.738$-0.934 between right and lefts sides of the body and $r = 0.563$-0.890 between observers. All correlations between observers were significant ($p<0.002$). The authors concluded that pressure threshold measurements between right and left sides of the body are highly reproducible.

Finally, a more recent investigation by Nussbaum and Downes (1998) examined three key issues in pressure algometry: (1) The reliability of pressure threshold measurements obtained with repeated measurements on consecutive days; (2) The reliability of PPDT measurements between examiners; (3) The number of measurements required to obtain the best estimate of PPDT.[162] Two examiners measured the pressure pain threshold of the biceps brachii on the non-dominant arm in 35 pain free volunteers. Participants were exposed to three measurements by each examiner on three consecutive days. A ten second interval was given between trials. A total of 18 measurements were obtained per subject. The intraclass correlation was calculated to estimate the inter-rater trial to trial and day to day reliability. For between examiner trials, the authors reported a range of intraclass correlations of 0.74-0.89 for single trials and 0.81-0.86 for multiple trials. All correlations were significant ($p<0.0001$). Inter-rater reliability improved when the mean of three daily scores was considered for data analysis instead of individual scores. Intraclass correlation coefficients for pressure threshold measurements recorded by the same examiner, trial to trial, ranged from 0.94-0.98. Measurements recorded on consecutive days by the same examiner showed intraclass correlation coefficients ranging from 0.88-0.90. All correlations were considered highly significant.

Validity and Clinical Utility of PPDT Measurement with PTM

The validity of pain intensity measurement is critical to the scientific quantification of pain. Measurement validity, considered to be the "appropriateness, meaningfulness, and usefulness of the specific inferences made from a test score", requires that PPDT measurements are not only reproducible but also valid.[163] Furthermore, research validity determines the extent to which test results are believable.[158] The primary obstacle to determining the validity of pain measurement is the inexistence of a gold standard comparison for PPDT measures. The evaluation

of pain can sometimes be confounded by emotional states (fear, depression, and hysteria), secondary gain, medication, gender, or concurrent pain conditions. These factors may lead to the misinterpretation of pain threshold measurements.

The pressure threshold examination has been used repeatedly in the determination of tenderness and hypersensitivity of various body regions. A study by Gunn and Milbrandt (1976) evaluated the pressure sensitivity of suspected motor points in 100 subjects with low back pain (50 subjects with 'low back sprain' and 50 subjects with 'disc involvement') and 100 control subjects (50 subjects with no history of low back pain and 50 subjects with occasional backache).[164] Researchers applied pressure, using their thumb, to 20 points on the lower extremity of each subjects and then graded the sensitivity at each point on a scale from 0-3 (least to most sensitive). The authors did not discuss the reliability of thumb pressure testing in measuring pain sensitivity. Their results showed that 52% of 'low back sprain' subjects and 98% of 'disc involvement' subjects reported tender motor points compared to 14% of 'no back pain' subjects and 92% of 'occasional backache' subjects. The authors suggested that motor point tenderness occurs most frequently in persons suffering from 'low back sprain' or 'disc involvement' compared to pain free individuals. As well, persons with 'low back sprain' and normal motor point tenderness showed a shorter duration of disability than those with 'low back sprain' and increased motor point tenderness. Researchers speculated that recovery time may be related to the degree of tissue injury, measured by pressure pain threshold testing.

A number of studies have emphasized the effectiveness of pain threshold quantification using the pressure threshold meter (PTM) in pain free subjects. Chung et al (1992) applied an electronic PTM to 13 muscles in the head and neck region of healthy pain free volunteers.[161] The authors recommended the use of a PTM to evaluate pain threshold in human head and neck muscles that tend to be accessible, flat, and supported by a hard underlying surface. Fischer (1987) tested the clinical applicability of the PTM in an attempt to develop normative values for pressure sensitivity over normal pain free muscles.[160] He studied the pressure pain detection threshold (PPDT) of 50 healthy individuals at nine typical trigger point sites in the paraspinal, proximal shoulder, and hip regions. He suggested that the PTM might be useful for measuring treatment success after therapeutic intervention (physical or pharmacological) or to objectify the exacerbation of painful conditions. The substantial difference between subject PPDT variations related to test site discovered in this investigation underline the importance of good clinical judgment when assessing the degree of pathological tenderness in persons who report pain. Another interesting PPDT study published by Hogeweg et al (1992) examined local tenderness at the wrist, elbow, knee, and ankle joints and paravertebral soft tissues of 28 pain free subjects.[120] The investigators suggested that pathological tenderness could be identified by comparing tender points to the same body region on the contralateral side, normative values in gender matched healthy adults, or to repeated measures within the same subject.

In an effort to improve the validity and reliability of pressure threshold measurement, Hardy et al (1952) suggested six PPDT measurement requirements. Their suggestions included: (1) Measurability of stimulus with reproducibility; (2) Controllability; (3) Adequate range from threshold to ceiling; (4) Production of minimal tissue damage; (5) Convenience; (6) Production of clear-cut pain perception.[165] The electronic PTM satisfies all six suggested criteria, and has been supported in the literature as an effective tool for the quantification of pain sensitivity in both healthy and painful subjects.

Reliability and Validity of Selected Pain and Disability Measurement Tools

Pain Drawings

A pain drawing is comprised of a line drawing, from the anterior and posterior perspective, of a non-gender specific person. The pain drawing provides an opportunity for subjects with pain to visually describe his or her pain experience to the investigator. Pain drawings are frequently employed for clinical and scientific purposes such as documenting symptom location, correlating physical data with psychological evaluations, determining changes in pain location, and predicting treatment outcome.[166]

The scoring technique for pain drawings has been a topic of controversy over the past number of years. One method of scoring, known as the penalty point system, has become increasingly popular due to its repeatability and strong correlation with psychological distress in chronic pain sufferers. The penalty point system, developed by Ransford et al (1976), assigns points for pain not thought to be typically associated with lumbar spine disease.[167] This system is based on the hypothesis that persons with poor psychometrics would show some or all of the following characteristics: (1) Unreal drawings showing poor anatomic location (i.e. bilateral foot pain); (2) Drawings showing "expansion" or "magnification" of pain (i.e. pain outside the body); (3) "I particularly hurt here" indicators (i.e. the use of arrows for emphasis); (4) "Look how bad I am" indicators (i.e. a tendency towards total body pain).[168] A drawing is considered normal if 2 or less points are assigned or as abnormal if the drawing scores higher than 2 points. High intra-rater reliability has been demonstrated with the penalty point system of scoring for pain drawings.[166,167] Ransford et al (1976) reported a overall association between the pain drawing and the Minnesota Multiplasic Personality Inventory (MMPI) of 89%. They also suggested that the pain drawing might be an approximate predictor of the Hs (hypochondrasis) and Hy (hysteria) scales of the MMPI. A biserial correlation of .60 (hypochondriasis - Hs) and .62 (hysteria - Hy) was reported between a score of greater than 70 (on Hs or Hy) and a pain drawing score of three or more. A total score of more than 70 on the Hs or Hy clinical scales is two or more standard deviations above the mean and occurs in only 2.5% of the normal population. They reported that penalty pain ratings of three or more occurred in 93% of subjects whose Hs or Hy score was greater than 70 and showed significant psychological involvement in their pain condition. Ransford et al (1976) suggested that a pain drawing may allow the physician to screen out patients with "poor

psychometrics" who may be more likely to have a poor result following orthopedic surgery.

Other studies have demonstrated the repeatability of the penalty point scoring system. Ohnmeiss (2000) investigated the reliability of scoring the pain drawing using the penalty point system in a population of low back pain subjects, and reported a κ value of 0.5884 suggesting high intra-rater repeatability.[166] Another study by Main (1984) evaluated the reliability of applying the penalty point system to scoring 100 pain drawings, and reported correlation coefficients of 0.77 for inter-rater reliability and 0.76 for intra-rater reliability.[169] The weakness of the penalty point system is that it requires subjective interpretation, by the examiner, to score the drawing.

Roland Morris Disability Index

Roland and Morris (1983) developed the Roland Morris Disability Index, for clinical and research settings, to measure self-rated disability amongst low back pain sufferers.[170] In its conception, the Roland Morris Disability Index (RMDI) was derived from low back pain related statements in the Sickness Impact Profile. The RMDI asks subjects to answer 24 "yes or no" questions and the number of "yes" answers are tallied to represent each individual subject's disability score (from 0 – no disability to 24 – severe disability). Roland and Morris examined the repeatability of this disability questionnaire in twenty low back pain sufferers on 2 consecutive days. Test-retest reliability scores showed a intraclass correlation coefficient ranging between 0.79 and 0.91, suggesting excellent reliability among low back pain sufferers.[171,172] Test-retest correlations have also been reported to range from 0.72 to 0.91 depending on the duration of time elapsed between repeated measures.[173,174] The validity of this questionnaire has been examined by comparing responses on the RMDI to responses on a six-point pain rating scale.[170] The Roland Morris Disability Index scores showed good agreement with the six point pain rating scale within 95% confidence limits. Other studies have demonstrated the accuracy of the RMDI to discriminate between two groups of low back pain sufferers with variations in pain severity as well as a modest correlation with physical function.[174,175]

Numeric Rating Scale

The numeric rating scale is a simple 11-point pain intensity measurement in which subjects are asked to rate their pain intensity with a number from 0 (no pain) to 10 (worse possible pain). The numeric rating scale can be administered with paper and pencil or over the phone and does not require subjects to have a high level of verbal or academic ability. The numeric rating scale also correlates well with other measures of pain intensity such as the visual analogue scale and the 'simple descriptive scale' yet the numeric rating scale is considered superior as it shows less measurement error.[176] In addition, the numeric ration scale has been showed to be sensitive in detecting changes in pain intensity in response to treatment.[177] Jensen et al (1994) discovered that "10 to 21-point scales provide sufficient levels of discrimination, in general, for chronic pain patients to describe pain intensity".[178] Test-retest correlations have been

reported to range from 0.67 to 0.96, suggesting good to excellent repeatability.[179,180,181] The numeric rating scale has become popular in clinical research as it is easy to administer, quick to score, and can be applied to a wide variety of subject groups.

Variables

Independent Variable:

Low back pain status (3 categories):

a. Acute low back pain (pain for less than three weeks),
b. Chronic low back pain (pain for greater than six months),
c. No low back pain (no low back pain in past six months)

Dependent Variable:

Pressure pain detection threshold (PPDT) – lbs/cm^2

Descriptive Variables:

A number of descriptive variables were recorded to characterize the subject groups. The descriptive variables included:

b. Age (years)
c. Duration of pain experienced – (converted to days, assuming a 30 day month),
d. Impact of low back pain on employment status (unemployed, alternative employment, modified duty employment, no effect on employment)
e. Disability Measure – The Roland-Morris Disability Scale describing each subjects' perceived level of disability (see Appendix H),
f. Pain Intensity –Numeric Rating Scale (see Appendix L),
g. Pain Drawing – scored with penalty point system.

Data Collection:

Independent Variable:

The independent variable was low back pain group designation including acute, chronic, and no low back pain. The pain duration experienced by each participant was recorded on the subject questionnaire and verified through a personal interview. Pain duration was measured in days for the 'Acute Low Back Pain Group' and number of months for the 'Chronic Low Back Pain Group'.

Pain duration was converted to *number of days* for all subjects. A 30-day month was assumed in the conversion calculation.

Dependent Variable:

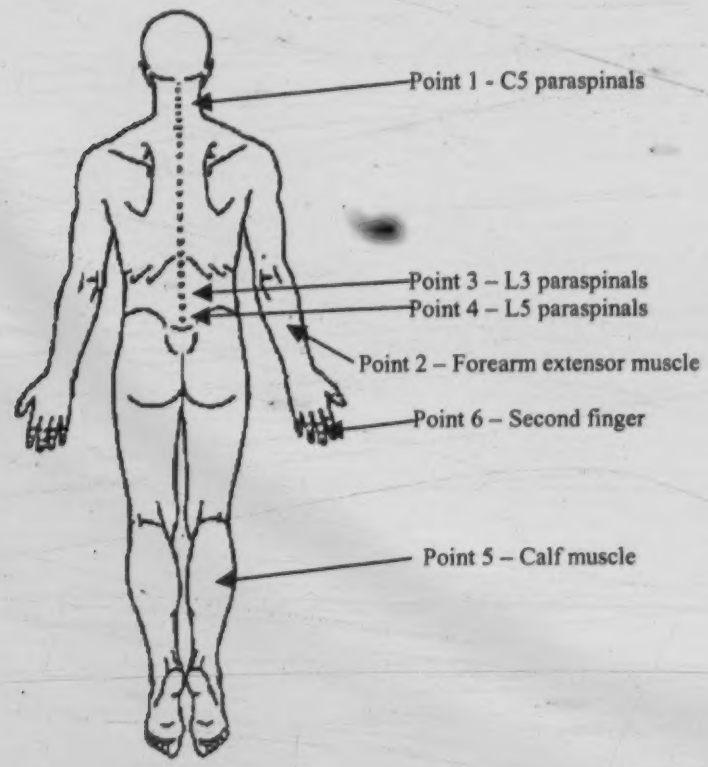
The pressure pain detection threshold (PPDT) was measured in lbs/cm^2 using a hand held electronic pressure threshold meter (PTM). An electronic PTM, commonly

used for PPDT measurement, was obtained from JTech Inc., Salt Lake City, Utah, USA. An independent assessor with anatomical knowledge was trained in the measurement technique. Training included discussions on pressure point location, use of the PTM (as per manufacturer's recommendations), restricted subject-examiner interaction and the method of recording pain threshold results. The measurement technique was the same for each subject. The assessor was blinded to the low back pain status of the subject being tested. The PPDT was dependent on the loading rate of the pressure and the size of the pressure area.[182] The applied method of testing was as described by Hogeweg et al (1992), a steady pressure rate of 2 lbs/sec with a pressure head surface area of 1 cm^2 and perpendicular alignment of the pressure threshold meter to the subject's skin.[93] The value of each point was the average of three test values. Ten seconds of recovery time was allowed between repeated measurements at each test site. Each test site was associated with a number (from 1 to 6) and the primary investigator used a random number generator to randomly select the order of test point measurement for each subject. The random order of test points was provided to the assessor just prior to testing each specific subject. The sites were numbered as follows: 1 = cervical, 2 = wrist extensor muscle, 3 = L3 paravertebrals, 4 = L5 paravertebrals, 5 = calf muscle, 6=middle phalanx of second finger (D3). The order of testing was attached to each subject's data sheet. Test results were not visible to patients. Bilateral testing was not imperative since previous studies did not show a significant difference in pain threshold between right and left sides when testing pain free subjects.[123,183,184] Yet, to ensure that global pain thresholds are thoroughly represented and that unilateral pain states are not accidentally omitted, all points were tested bilaterally.

Prior pressure threshold research has demonstrated considerable inconsistency and variability in the selection of test sites for the examination of subjects with low back pain. Clauw et al (1999) investigated the pain sensitivity levels of chronic low back pain patients as a correlate of functional status.[58] Researchers chose to investigate 18 common tender points throughout the body (the specific points not indicated in the study) and four control points. Peters, Schmidt and Van den Hout (1989) studied the reaction of chronic low back pain patients to acute pain stimulation with a PTM.[149] Six trials of pain stimulation were recorded from the middle phalanx of the second digit on the non-dominant hand. Rationale for test site choice was not provided. A few years later, Peters and Schmidt (1992) tested and compared pain sensitivity levels between chronic low back pain patients and healthy pain free subjects.[185] A single test point at the middle phalanx of the second digit on the non-dominant hand was chosen. The researchers again did not provide rationale for their test site selection. Another group of researchers studying the effects of spinal manipulation on PPDT in chronic low back pain subjects chose three test sites in the low back region including L5 paraspinals, sacroiliac ligament and the gluteal muscles.[186] Pre and post treatment measurements (4 in all) were recorded at each location. Test points were selected based on their relationship to mechanical low back pain and consistency with typical myofascial trigger point locations. In studies of the thermal pain thresholds in low back patients, researchers have commonly selected test sites in the hand and forearm regions.[212,187,188]

Twelve test sites (six per side) were chosen for this study (see Figure 1). The 6 test sites, measured bilaterally for each subject, included: the paravertebral region 2cm lateral to L5 and L3, paravertebral region 1 cm lateral to C5, the wrist extensor muscle belly, the calf muscle belly and the middle phalanx of the second digit. Test point locations were chosen to ensure all body regions were adequately represented in PPDT measurements and to avoid common trigger point sites to ensure that pressure threshold measurements were not skewed by small areas of hypersensitivity. In all subjects, test points were not located on an open wound or area of intense swelling or bruising. A test point on the middle phalanx of the second digit was selected to improve comparability of this study to other investigations of pressure pain threshold in chronic low back pain subjects using this test site.

Figure 1: **PPDT Test Sites (tested bilaterally)**


Point 1 - C5 paraspinals
Point 3 – L3 paraspinals
Point 4 – L5 paraspinals
Point 2 – Forearm extensor muscle
Point 6 – Second finger
Point 5 – Calf muscle

Descriptive Variables

Data were collected for descriptive variables through a self questionnaire package, completed by each subject following the interview with the primary investigator and completion of the consent form. The questionnaire package included the Roland Morris Disability Index, a Pain Drawing, a Numeric Rating Scale, and a demographic questionnaire.

Procedure

This experiment took place in a soundproof room of constant temperature between the hours of three and six p.m. Each test session lasted approximately 30 minutes. The primary investigator interviewed each subject to confirm that all inclusion criteria had been satisfied prior to testing, and discussed the ethical considerations of the research project with each subject. Participants were asked to complete a consent form (see Appendix B) and a subject questionnaire (see Appendix D). The subject questionnaire requested information on age, employment status, medication use, disability, and pain location. Each subject was asked to complete a Pain Drawing (see Appendix M), a Numeric Pain Rating Scale (see Appendix L), and the Roland Morris Disability Questionnaire (see Appendix H).[170,189,190,191]

Each participant received a verbal explanation of the test procedure along with a subject information sheet (see Appendix E). The primary investigator conducted a preliminary 'test run' on each participant's thigh, while in sitting, to familiarize the subject with the measurement technique. The definition of pressure pain detection threshold was reviewed and reinforced. The primary investigator ensured that each subject clearly understood that the PPDT was achieved when pressure became painful.[122] Following the completion of all forms, the interview, and the 'test run' with the PTM, the primary investigator introduced each subject to the research assistant performing the examination without revealing low back pain status. Participants were asked to assume a prone or side lying on the examining table for the entire testing process unless a position change was required to relieve low back pain exacerbation. Subjects were given the option of placing a pillow under their pelvis for comfort. The research assistant then revealed the random test site selections and marked each test point on the right and left sides of subject's body with a black felt pen. Right and left side test sites were tested in the same sequence. The assessor tested each point three times beginning on the right side and alternating between right and left sides. The average of three measurements represented the average PPDT at that point. The PTM was applied in a perpendicular position to the subject's skin and pressure was increased at a rate of approximately 2 lbs/sec as the examiner observed the rate of pressure application on the screen of the pressure threshold meter.[93,117,160] The subject was asked to indicate the PPDT by stating "pain". The PTM records the highest pressure reading after the applicator has been withdrawn. The PTM calculated the mean value for each point measured (based on three measurements) and then saved all PPDT measurements to be transferred later to each subject's data sheet (see Appendix J). The subject was instructed that she was free to stop the examination for any reason, particularly if she wanted to ask a question about the examination, or was uncomfortable with the testing procedure. The primary investigator met the subject after the test to answer any final questions and provide the subject with a card of thanks with $10 remuneration enclosed. The subject was asked to avoid discussing the test procedure with other subjects who may have been waiting for testing. All data was entered into a computerized spreadsheet for data analysis after testing was complete on each day.

Data Analysis

Considering the difficulties associated with the recruitment of acute low back pain sufferers, only chronic low back pain sufferers and pain free subjects were included in the data analysis. Six acute low back pain sufferers were recruited and tested using the methods described above. A sample size of six allows for only limited generalizability and risks an elevated Type I error when inferences are drawn from the subject group to the population. Thus the inability to recruit sufficient numbers of acute low back pain subjects resulted in the deletion of the 'Acute Low Back Pain' subject group from the primary and secondary hypotheses. This investigation became a two-arm study, comparing chronic low back pain sufferers with pain free subjects. In light of this alteration in the design of the study, analysis of variance and Tukey's pairwise comparisons were no longer required in the inferential statistical analysis. A simple t-test was applied to compare group means. An alpha level of .05 was employed as the level of significance.

This investigation presently examines the following primary and secondary hypotheses.

1. Primary Hypotheses:
    a. Chronic low back pain sufferers will demonstrate a lower global PPDT compared to pain free volunteers.
        i. Null Hypothesis: No difference in pain threshold exists, at the distal test sites, between chronic low back pain sufferers and pain free subjects.
    b. Chronic low back pain sufferers will demonstrate a lower PPDT at sites related anatomically to the low back region compared to pain free subjects.
        i. Null Hypothesis: No difference in pain threshold exists, at sites related anatomically to the low back region, between chronic low back pain sufferers and pain free subjects.
    c. Chronic low back pain sufferers will demonstrate a lower pain threshold at sites unrelated to the low back region compared to pain free subjects.
        i. Null Hypothesis: No difference in pain threshold exists, at sites unrelated to the low back region, between chronic low back pain sufferers and pain free subjects.

2. Secondary Hypotheses:
    a. Higher Pain Drawing scores will be associated with lower global PPDT scores in chronic low back pain subjects.
        i. Null Hypothesis: No correlation will exist between pain drawing score and global pain threshold in chronic low back pain subjects.

Descriptive Statistics

Descriptive statistics were analyzed differently depending on the type of variable. Continuous variables were summarized using mean, standard deviation, and percentage. Discrete variables were summarized using frequency and percentage.

Inferential Statistics

In this study, independent t-tests were applied to compare mean values between groups. Global pain threshold, represented by the entire set of test sites, was compared between subject groups. Test sites were also divided into groups to represent two body regions to allow for comparison between subject groups. Regional test site groups included: (1) test sites unrelated to the lumbar spine (finger, forearm, and neck); (2) test site anatomically related to the lumbar spine (L3 paraspinals, L5 paraspinals, calf muscle). The mean value of point 6 (finger) was also compared between groups.

Ethical Considerations

This study was approved by the University of Alberta Health Ethics Research Board (panel B) and the Bioethics Committee of the Lloydminster Hospital.

Pressure pain detection threshold is a measurement that allows clinicians or researchers to quantify tenderness in patients and/or subjects. Despite the presence of a normal distribution of inter-individual differences in pain threshold, pressure threshold measurements are clinically relevant for identifying differences between groups or changes in pain threshold following repeated measures of the same group. This type of research is important for gathering normative data in pain free subjects as well as identifying alterations in the distribution of tenderness or hypersensitivity in persons experiencing pain. The benefits of this study are mostly accrued to future subjects. The main purpose of this research is to improve assessment techniques presently applied to evaluate persons with low back pain. Therefore, subjects who undergo testing do not receive an intervention designed to reduce their pain. By choosing to test pain threshold instead of pain tolerance, subjects will not experience and exacerbation or worsening of their pain condition. The risk of harm was minimized in the design in a number of ways: (1) Test sites were selected in areas of soft tissue and typically not tender to pressure; (2) Subjects were allowed to lie in any position for testing and encouraged to sit, stand, or walk at any point during the test procedure to relieve discomfort;. (3) Areas of obvious inflammation or open tissue were avoided; (4) A minimal number of sites were tested to achieve the goals of the study; (5) Subjects were instructed to stop testing and ask questions at any time and were allowed to discontinue participation in the project without negative consequences.

Burdens or inconveniences of the research were reduced in a number of ways. Subject screening interviews were conducted by telephone, whenever possible, to minimize unnecessary travel required by each potential subject. Also, the data collected through questionnaires was streamlined to minimize the amount of

paperwork required by each participant. The amount of time required to complete the forms and pain threshold testing was condensed to 30 minutes and subjects were only required to visit the clinic once. The inconvenience of travel was reduced by holding the project at a familiar location with an ample amount of free parking. Other burdens such as childcare and fuel costs were offset by offering each subject a modest remuneration of $10. The principle investigator was also a clinician and did not directly recruit his own low back pain patients for participation in the research. In this way, potential subjects did not feel obliged or coerced into participation.

Participants in this study did not report any adverse effects from pain threshold testing. A contact person from the Faculty of Graduate Studies and Research and the Health Research Ethics Board was provided to each subject if a concern arose about the research.

Results

Descriptive Characteristics of Study Subjects

Thirty chronic low back pain and thirty pain free subjects were included in this analysis. The mean ages of chronic low back pain and pain free subjects were 41.6 ± 9.7 years (range: 20-59 years) and 42.2 ± 9.5 (range: 24-59 years) years respectively. Chronic low back pain subjects reported a mean(SD) pain duration of 11.6(9.2) years and a mean(SD) numeric pain rating score of 5.9(1.8) out of 10. Disability scores on the Roland Morris Disability Index ranged from 0-20 with a mean(SD) of 7.5(5.0) out of 24. Seventy percent of chronic low back pain subjects reported that low back pain had no effect on their function and 37% of chronic low back pain subjects used medication for symptomatic relief. Only 10% (n=3) of chronic low back pain subjects scored 3 or more on the pain drawing, such that associations between abnormal pain drawings and global PPDT scores could not be examined. Additional descriptive characteristics of the chronic low back pain group are provided in Table 2.

Table 2: **Pain Location – Chronic Low Back Pain Group**

	Left LBP	Right LBP	Left + Right LBP	Central LBP	Left Leg Pain	Right Leg Pain	Bilateral Leg Pain	No Leg Pain
CLBP Group	13%	27%	33%	27%	13%	20%	10%	57%

Pain free subjects did not report pain in any body regions. Scores on the Numeric Rating Scale, Roland Morris Disability Index, and Pain Drawing were zero for all pain free subjects.

No individuals referred to the study who met inclusion criteria were excluded. However, the number of persons seen by clinicians at the participating referral clinics who would have been eligible for the study, but were not informed of the study or chose not to participate, is not known.

Inferential Analysis and Hypothesis Testing

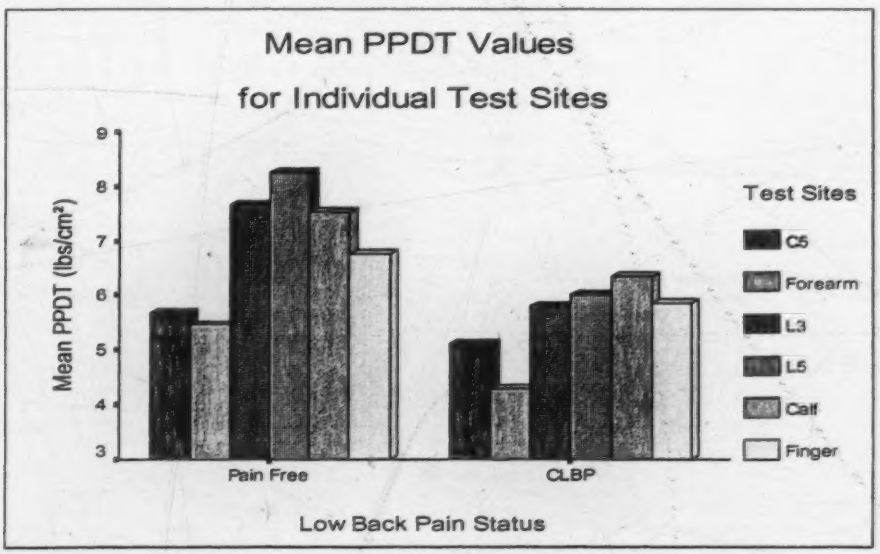
Individual test site mean PPDT values and t-test comparisons are summarized in Table 3 for pain free subjects and chronic low back pain subjects. Mean PPDT values showed a tendency towards being greater at each test site in the pain free group as compared to the chronic low back pain group but not all differences were statistically significant. Graphical representation of each test site is provided in a bar graph (Figure 2).

Table 3: **Mean (SD) PPDT Values and t-test Comparisons at Each Test Site**

	C5 paraspinal	Wrist extensor	L3 paraspinal	L5 paraspinal	Calf muscle	2nd finger
Pain Free	5.7(3.5)	5.5(2.0)	7.7(2.9)	8.3(3.2)	7.6(2.4)	6.8(1.9)
CLPB	5.1(3.2)	4.3(1.9)	5.8(3.1)	6.1(3.1)	6.3(2.6)	5.9(1.7)
t-value	0.65	**2.33***	**2.39***	**2.76***	1.90	1.95
Sig (2-tail)	.52	**.02**	**.02**	**.01**	.06	.06

all measurements are in lbs/cm²
(n = 30, *p < .05, two tails

Figure 2: **Mean PPDT Bar Graph Representation by Individual Test Site**



Test sites were grouped to represent body regions for analyses. Test sites at the L3 paraspinals and L5 paraspinals were grouped to represent sites anatomically *related* to the low back region. Test sites at the C5 paraspinals, wrist extensor muscle, and finger were grouped to represent sites that were anatomically *unrelated* to the low back region. The global PPDT was considered to be all six points combined. The finger test site was provided in isolation for comparison with other studies. All test sites were measured bilaterally. A summary of the mean PPDT values for each grouped body region is given in Table 4. Graphical representation is provided in Figure 3. Mean PPDT values, representing test site combinations, were compared between pain

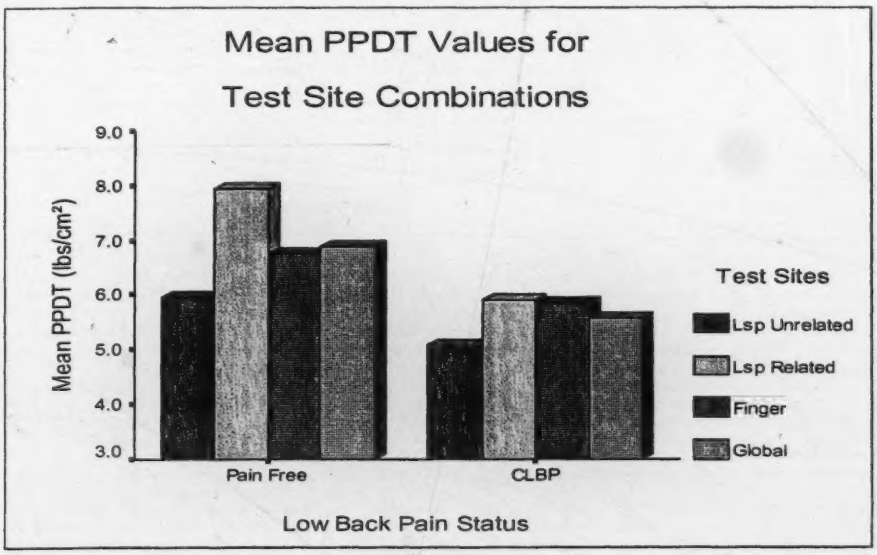
free and chronic low back pain groups. An independent sample t –test (df=28) was applied to determine whether a difference existed between grouped means ($p<.05$). The combined mean PPDT comparison between chronic low back pain and pain free groups is summarized in Table 4.

Table 4: **Mean (SD) Combined PPDT Values and Summary of Combined Mean PPDT Analysis for Pain Free and Chronic Low Back Pain Groups**

	Global PPDT	Lsp Unrelated PPDT	Lsp Related PPDT	Finger PPDT
Pain Free	6.9(2.1)	6.0(2.0)	8.0(2.9)	6.8(1.9)
CLBP	5.6(2.1)	5.1(1.9)	5.9(3.0)	5.9(1.7)
t-value	**2.45***	1.72	**2.73***	**1.95**
p-value	0.02	**0.09**	0.01	**0.06**

(n = 30, *p < .05, two tails)
all measurements are in lbs/cm²

Figure 3: **Grouped Mean PPDT Bar Graph Representation**



Comparison of All Test Points (Global Mean PPDT)

The global mean PPDT value, encompassing all test sites, was calculated at 5.6 ± 2.1 lbs/cm² for chronic low back pain sufferers and 6.9 ± 2.1 lbs/cm² for pain free subjects. An independent sample t-test comparison of means resulted in a t-value of 2.45 with a p-value of .02. The null hypothesis, which states that a difference in global pain threshold *does not exist* between chronic low back pain subjects and pain free subjects, *was rejected* at .05 alpha level ($.95t(58) = 2.45$, $p<.05$).

Comparison of Test Sites Anatomically Unrelated to the Lumbar Spine

Test points combined for this analysis included the right and left sides of: test sites at the C5 paraspinals, wrist extensor muscle, and the middle phalanx of the second finger. Chronic low back pain sufferers demonstrated a mean PPDT of 5.6 ± 2.0 lbs/cm². Pain free subjects showed a mean PPDT of 5.1 ± 1.9 lbs/cm². In comparing the two groups, the resultant t-value was 1.72 and the p-value was .09. The null hypothesis, stating that a difference in the PPDT at sites unrelated to the lumbar spine *does not exist* between chronic low back pain and pain free groups, *could not be rejected* at .05 alpha level(.95t(58)=1.72,p<.05).

Comparison of Test Sites Anatomically Related to the Lumbar Spine

Test points combined for this analysis included right and left sides of: test sites at the L3 paraspinals and L5 paraspinals. Chronic low back pain sufferers demonstrated a mean PPDT of 5.9 ± 3.0 lbs/cm² and pain free subjects showed a mean PPDT of 8.0 ± 2.9 lbs/cm². An independent sample t-test was applied to determine whether a difference existed between these two groups of subjects. A t-value of 2.73 with a significance value of .01 was recorded. Using an alpha level of .05, it was determined that it was likely that the difference between the means of chronic low back pain and pain free subjects was a true difference and not a product of chance. The null hypothesis, stating that a difference in PPDT at the low back region *does not exist* between chronic low back pain and pain free groups, *was rejected* at .05 alpha level (.95t(58) = 2.73, p<.05).

Single Point Comparison at the Middle Phalanx of the Second Finger

A comparison of the mean pressure pain detection threshold at test site #6 (second finger) on both hands was completed between pain free and chronic low back pain groups. The mean PPDT was calculated at 5.9 ± 1.7 lbs/cm² for chronic low back pain subjects and 6.8 ± 1.9 lbs/cm² for pain free subjects. An independent sample t-test comparison produced a t-value of 1.95 and a significance value of .056. The null hypothesis, which states that a difference in PPDT at the second finger *does not exist* between chronic low back pain and pain free groups, *could not be rejected* at .05 alpha level (.95t(58) = 1.95, p<.05).

Discussion

In this study, pressure threshold testing was undertaken in order to identify differences in pain sensitivity between chronic low back pain sufferers and pain free subjects. It was hypothesized that chronic low back pain sufferers would demonstrate lower pressure pain detection thresholds in all body regions tested, in body regions related to the lumbar spine, and in body regions unrelated to the lumbar spine.

Global pain threshold was considered to be the mean value representing all six test sites examined bilaterally in each subject. Global pain threshold values were compared between chronic low back pain sufferers and pain free subjects. This

analysis revealed a lower global PPDT in chronic low back pain subjects compared to pain free volunteers. This result suggests that chronic low back pain subjects were generally more sensitive to pressure than pain free subjects. Pressure pain detection threshold measurements tended to be lower in chronic low back pain sufferers than pain free subjects at all test sites. A lower global PPDT identified in chronic low back pain sufferers may be related to alterations in the central nervous system and maladaptive pain processing. It is also likely that biopsychosocial influences may have contributed to widespread hypersensitivity. Unfortunately, the presence of support for the latter could not be examined through investigating associations between PPDT and pain drawing scores because too few subjects had abnormal pain drawing scores.

Following the comparison of global pain threshold, test sites were separated into two sections: (1) Body regions neuroanatomically *related* to the low back (L3 paraspinals, L5 paraspinals, and calf muscle); (2) Body regions NOT neuroanatomically related to the low back (C5 paraspinal, wrist extensor muscle, and finger). These body regions were compared separately between chronic low back pain and pain free groups for differences in mean PPDT. Measurements obtained bilaterally from the test sites at the L3 paraspinals and L5 paraspinals demonstrated a significantly lower pain threshold in chronic low back pain sufferers compared to pain free subjects. This result was in agreement with the generally accepted clinical assumption which suggests that chronic low back pain sufferers tend to report hypersensitivity to pressure applied to the low back region compared to pain free subjects. The results of this study may also suggest an element of persistent peripheral sensitization involving local hypersensitivity at the low back region in chronic low back pain sufferers.

The comparison of neuroanatomically *unrelated* test sites (C5 paraspinals, wrist extensor muscle, and finger) between chronic low back pain and pain free groups revealed a lower pain threshold in chronic low back pain sufferers compared to pain free subjects yet the difference was not statistically significant ($p<.05$). Chronic low back pain sufferers demonstrated a significantly lower pressure pain threshold at the wrist extensor muscle belly but not at the second finger or the cervical paraspinals. This result suggests two possibilities: (1) Chronic low back pain sufferers did not show significant widespread changes in pain sensitivity; (2) Or if widespread changes in pain threshold did occur, the approach to pain threshold testing was not adequately sensitive to detect alterations in pain sensitivity. It has been suggested that widespread pain or secondary hyperalgesia is often demonstrated when testing pain sensation with punctuate or light touch stimulus (Aβ-fibers) versus blunt pressure (C-fibers) applied by the pressure threshold meter.[192,193] Central sensitization may be more involved in mediating impulses from non-nociceptive Aβ-fibers than from C-fiber nociceptors.[194] The pressure threshold meter may be well suited in detecting the peripheral sensitization of C-fibers and primary hyperalgesia but unable to identify the central sensitization of input from non-nociceptive Aβ-fibers. In this case, widespread pain or pain in adjacent uninjured tissues would persist undetected if testing performed with a blunted tipped pressure threshold meter.

The difference in global pain threshold evident between groups may be a result of differences at the low back region and not global alterations in pain threshold. Pain threshold measurements from the L3 paraspinals and L5 paraspinals could have influenced the global PPDT more than other sites, suggesting that the inference of global hypersensitivity might be erroneous. This would be unlikely since mean PPDT values at the wrist extensor muscle, L3 paraspinals, and L5 paraspinals were significantly lower in the chronic low back pain group compared to pain free subjects. Pain threshold measurements at the calf muscle and finger were nearly significant (p-value of 0.064 and 0.056). The only test site where the difference in mean PPDT was not clearly dissimilar between groups was test site C5 paraspinals. A definite tendency towards generalized hypersensitivity was evident in chronic low back pain sufferers compared to pain free volunteers. Moreover, the mean PPDT at all test sites was lower in chronic low back pain sufferers compared to pain free subjects.

Previous studies have confirmed the presence of hypersensitivity and decreased pressure pain threshold values in regions that are reportedly painful. Subjects with Rheumatoid Arthritis, Osteoarthritis, and Juvenile Chronic Arthritis show a significantly lower PPDT, at the affected joints, compared to healthy adults.[195,196,197,93] Persons suffering with neck pain resulting from traumatic injury or office related postural stress, demonstrate local hypersensitivity and a lower pressure pain threshold at points tested in the cervical region than pain free subjects.[198,199] Chronic tension-type headache sufferers also tend to show decreased pressure pain detection thresholds in the pericranial myofascial tissues, as compared to pain free subjects, suggesting the possible role of peripheral sensitization in their pain experience.[200,201,202,77]

Peters, Schmidt, and Van den Hout (1989) conducted a pain threshold investigation involving chronic low back pain subjects and pressure pain stimulation.[149] Forty age-matched male participants (20 pain free and 20 chronic low back pain subjects) underwent pain threshold testing with a Forigone-Barber pressure stimulator on the middle phalanx of the second finger for six stimulation trials. The results of the study showed that chronic low back pain sufferers had a higher PPDT than pain free subjects but the difference between groups was not statistically significant. Peters and Schmidt (1992) again studied the pain threshold of 20 chronic low back pain sufferers compared to 23 pain free subjects by collecting pain threshold measurements from the middle phalanx of the second finger of all subjects.[185] The researchers reported that chronic low back pain sufferers were less sensitive to pressure at the middle phalanx of the second finger than pain free control subjects. The authors suggested further that chronic low back pain sufferers tend to be *generally less sensitive* to pressure than pain free subjects. Chronic low back pain subjects showed significantly higher pain threshold and pain tolerance levels, at the second finger, compared to pain free subjects but no difference was found between groups in the pain discrimination task. Both studies recruited subjects through a newspaper advertisement in a local newspaper.

In the present study, a comparison of pressure pain threshold measurements recorded at the middle phalanx of the second finger showed that chronic low back pain sufferers tend to demonstrate a lower pain threshold than pain free subjects, but the

difference between means was not statistically significant ($p<0.05$). When global pain threshold, representing 12 points throughout the body, was considered, the present study contradicts Peters, Schmidt, and Van der Hout (1989) and Peters and Schmidt (1992) by suggesting that chronic low back pain sufferers are generally more sensitive than pain free subjects. The present study involved a greater number of test sites, local and distal to the lumbar spine, and may have been more sensitive in detecting alterations in PPDT than examinations involving only one test site. As well, the present study examined the PPDT of subjects who were health care seekers whereas, in the previous two studies, subjects were recruited through a newspaper advertisement. It is possible care seeking chronic low back pain subjects may vary in their perception of experimental pain and their attitude towards pain stimulation compared to non-care seekers.

A recent investigation by Johansen et al (1999) examined the pressure pain detection threshold of chronic whiplash sufferers at test sites within and outside the area of trauma.[14] This study was retrospective and examined a small number (n=11) of chronic pain subjects having pain for more than one year. The authors reported significant muscular hyperalgesia in the neck, shoulder, and tibial regions of chronic whiplash subjects compared to pain free control subjects. In this investigation, test sites were not combined to represent global pain threshold yet each individual test site was significantly more sensitive to pressure in the chronic whiplash group compared to pain free subjects. Essentially, this investigation agrees with the present study, suggesting that chronic pain sufferers may show global pressure hypersensitivity compared to pain free volunteers.

A prospective study by Kasch, Stengaard-Pedersen, et al (2001) investigated the pain thresholds of 123 acute whiplash patients and 36 control subjects in the head and neck region with a distal control site on the left finger.[203] Pain threshold measurements were taken at 1 week, 1 month, 3 months, and 6 months. Chronic whiplash sufferers showed marked hypersensitivity at all head/neck sites until 3 months but no significant difference at 6 months. Pressure pain detection threshold values measured at the distal control site (finger) were similar between groups at all examination intervals. In this study, control subjects were not pain free. The researchers selected a control group with a different acute pain condition in order to control for the potentially confounding effects of post-traumatic stress. The results of this study suggest that peripheral sensitization at the head and neck region is likely temporary and hypersensitivity of tissues unrelated to the area of injury may not occur. The authors reported significantly lower PPDT scores in female subjects from both groups but gender was not controlled in the design. This study contradicts the present investigation yet it is most likely that the samples of chronic pain sufferers were quite dissimilar. Factors that may contribute to this dissimilarity may include age, injury acuity, gender, involvement in disability insurance, and medication usage.

The results of the present study do not support the theory that 'pain inhibits pain' or the notion of 'adaptation' in chronic low back pain sufferers as suggested by previous studies.[185,204] The 'pain inhibits pain' theory suggests that if persons experiencing ongoing persistent pain are exposed to an experimental pain stimulus, the

two concurrent pain experiences form a noxious inhibitory mechanism. This noxious inhibitory mechanism results in decreased sensitivity and an increased pain threshold. The results of this investigation also contradict the theory of adaptation. The adaptation theory of pain suggests that chronic pain sufferers frequently compare a novel or experimental stimulus with their present state of pain. As such, chronic pain sufferers might consider an experimental pain stimulus to be relatively mild in the context of a greater persistent pain state. In the present investigation, chronic low back pain sufferers tended to show local (low back region) and global decreases in PPDT compared to pain free subjects. As well, the data showed a definite trend towards a lower PPDT in chronic low back pain subjects at all sites compared to pain free subjects but not all differences were statistically significant.

Clinical Implications

External stimulation exerted by the blunt pressure of a 1 cm^2 rounded contact surface activates nociceptive afferents in deep tissues (dermis, muscle, periosteum) more than cutaneous afferents.[205] Pressure on the skin alerts the organism to noxious mechanical stimulation or impeding external injury.[206] Pressure applied to the tissues of a pain sufferer is intended to provide information on tissue irritability or the presence of trigger and tender points. Pain sensitivity and tenderness are related to clinical status but also tend to be significantly associated with reported physical function.[58] Variations in PPDT have been shown to occur following injury or disease and tend to change in relation to a patient's condition.[112]

The present study identified that chronic low back pain sufferers tend to demonstrate a lower pressure pain detection threshold levels in the low back region compared to pain free subjects. At the onset of treatment, clinicians might obtain PPDT measurements from the low back region of a chronic low back pain sufferer and then retest the patient at specific intervals in order to track changes in pain sensitivity through the course of treatment. It is not known whether pain threshold values increase with improvements in subjective pain complaints in the chronic low back pain sufferer. Further research is required to determine if pain threshold measurements may be useful in quantifying the success of a physical or pharmacological intervention for the chronic low back pain sufferer.

The purpose of this study was to determine whether differences in pressure pain detection threshold exist between chronic low back pain sufferers and pain free subjects. It was suggested that a difference in pain threshold between these groups may help clinicians to better understand the pain presentation of chronic low back pain sufferers. This study supports the notion that persons with chronic low back pain are more generally more sensitive to pressure than pain free subjects. When chronic low back pain and pain free groups were compared for differences in PPDT at test sites unrelated to the lumbar spine, the results did not reach statistical significance. Nonetheless, a tendency towards decreased pain threshold and hypersensitivity was evident at all points tested in chronic low back pain subjects. Chronic low back pain sufferers who participated in this study were not screened for specific diagnoses or included in the study based on specific anatomical features. Rather, low back pain

subjects were recruited based on their subjective reports of pain. It may be useful for clinicians to appreciate that chronic low back pain sufferers tend to demonstrate generalized hypersensitivity to pressure in the presence of persistent pain rather than a specific diagnosis. Clinicians may also consider that persons suffering with chronic low back pain could be sensitive to pressure in regions that are not anatomically related to the low back region.

This investigation may challenge a purely physiological approach to the assessment and management of chronic low back pain. Goldsmith and Wiesel (1998) argued that a thorough history and physical examination of the chronic low back pain sufferer can result in an accurate diagnosis and treatment.[207] Katz (1993) suggests a simple classification that categorizes low back pain in terms of clinical features and associated physiological lesions.[208] He suggests that the clinical features associated with chronic low back pain may change as pain persists and the pain sufferers may become disassociated from physical findings and more connected to psychosocial disturbances. Waddell, Somerville, et al (1992) investigated the physical impairments of subjects with chronic low back pain and developed a method of routine clinical evaluation for this group of pain sufferers.[54] The authors reported that in regards to chronic low back pain, "...it is not possible to identify any objective pathology or reach a definite diagnosis". Furthermore, they suggest that the response of chronic low back pain sufferers to most clinical examination techniques will depend on pain-inhibition, fear-avoidance, psychological distress, and illness behavior in addition to physical impairment. Pain in regions unrelated to the lumbar spine may point toward maladaptive pain processing or the influence of psychosocial factors in a pain sufferer's clinical presentation.

Limitations

This investigation was designed to avoid the methodological hazards often associated with pain threshold research and control some of the factors that might threaten internal validity. Despite efforts to avoid experimental bias and resolve the influence of confounding factors, some aspects of this study could not be fully controlled. It is likely that aspects of this study that could not be fully controlled but were similar for both pain free and CLBP groups did not effect the results.

Verbal subject-examiner delay may have affected the accuracy of the test procedure. Subjects undergoing pain threshold testing were required to subjectively interpret the 'point at which pressure becomes painful' and then verbalize this moment to the examiner. Pressure applied by the examiner was immediately discontinued and the pressure threshold meter records the PPDT. Two opportunities are apparent for a delayed measurement of PPDT. First, the subject may perceive pain but delay verbalizing a response to the examiner. Second, the examiner may delay the discontinuation of pressure after hearing the subject's verbal response of pain. It seems reasonable that the first instance may be more plausible than the second. Subject-examiner delay would result in an inaccurately high pain threshold recorded in the individual subject. However, for verbal subject-examiner delay to affect the

comparison between chronic low back pain and pain free subjects, a difference in this delay error would need to be present between groups. This is not expected.

Examiner error and bias were obvious methodological pitfalls requiring special attention in the design and implementation of this research project. The observed differences in PPDT between painful and pain free sites may have been biased by examiner expectancy expressed through subtle alterations in applied pressure rate. In this study, the examiner was blinded to the low back pain status of each participant. In a similar way, PPDT measures may have been affected by alterations in examiner performance since the examination period lasted for more than a year. Through the duration of the study, the examiner was required to regularly review the test procedure. Particularly, if there was a pause in the recruitment and testing of subjects. Examiner fatigue was also an issue when more than three subjects were tested consecutively. The examiner reported that a constant rate and intensity of stimulus application became more difficult more than three consecutive subjects were tested. In order to prevent fatigue, a maximum of three subjects were tested consecutively on any given day. It is also difficult for the examiner to provide a slow constant increase in pressure at 2.2lbs/sec, particularly when subjects show an unusually high pain threshold. In order to accomplish constant pressure, the examiner required time to practice applying the pressure threshold meter and operated the unit with two hands at all times.

In some instances, consecutive PPDT measurements at the same test site demonstrated a trend towards progressive decreases in pain threshold. This measurement error may be related to temporal summation occurring as a result of insufficient recovery time allowed between repeated measures. Nociceptors may have still been sensitized from previous measurements when the next measurement was taken. A standard recovery period between measurements does not exist in the literature. In this study, a 10 second pause was built into the testing procedure to minimize the potential effects of temporal summation.

Inter-subject variability is an unavoidable limitation of pressure algometry. Natural variability in pain threshold exists normally in the general population.[160] In this study, sample size calculations were determined based on normative data (see Appendix A). Adequate sample sizes were achieved to reduce the effects of variability in the general population. In addition, subjects in the present study are quite similar to the chronic low back pain subjects recruited for other pain threshold investigations. Chronic low back pain subjects, in the present study, reported a mean (SD) age of 41.57(9.69) years and a mean (SD) pain duration of 11.55(9.23) years. Peters, Schmidt, and Van den Hout (1989) reported that the participating chronic low back pain subjects in their study reported a mean age of 41 years and a mean duration of back pain of 11.5 years.[149] Peters and Schmidt (1992) studied a group of chronic low back pain sufferers with a mean age of 42 years and a mean pain duration of 14 years.[185] Also, Lautenbacher, Galfe, et al (1990) measured pain threshold in a group of chronic low back pain sufferers with a mean age of 43.2 years and a mean pain duration of 10.3 years.[187] The sample of chronic low back pain subjects recruited for the present investigation appear similar to previous studies on chronic low back pain

sufferers allowing for acceptable comparability between studies. The similarity demonstrated between the groups of low back pain studies noted above also supports the notion that subjects studied in the present investigation may be representative of a larger population of healthcare seeking chronic low back pain sufferers.

Chronic low back pain sufferers who participated in this study were not recruited or excluded based on specific low back pain diagnostic criteria. Instead, this study was focused on identifying alterations in pain sensitivity in association with persistent pain potentially stemming from multiple physiological sources. Although a number of theoretical constructs allude to a physiological origin of chronic low back pain, the vast majority of chronic low back pain is idiopathic and the physiological source of chronic low back pain remains elusive. Thus, one of the limitations related to this investigation was that alterations in pressure pain detection threshold were not tested in subjects with a specific known physiological condition.

Recent research has demonstrated a relationship between pain threshold and the menstrual cycle. In a community cohort of 299 women, Angst et al (2001) reported a prevalence rate of 8.1% for severe and 13.6% for moderate premenstrual and menstrual symptoms.[209] In a similar fashion, Logue and Moos (1986) reported a prevalence rate of severe perimenstrual symptoms at 2-10% of females. Isslee et al (2002) evaluated the influence of the menstrual cycle on pressure pain threshold in 10 women with masticatory muscle tenderness.[210] The authors reported that during the follicular phase (day 10-12) and luteal phase (day 24-26), the pain threshold of all muscle sites increased by 16% to 42%. Pain threshold during the perimenstrual period (day 27, 28, and 1) was 10% to 30% lower than the pain threshold recorded in the luteal and follicular phases. Isselee et al (2001) also studied the influence of the menstrual cycle on pain threshold in a group of 10 pain free females.[211] The authors also reported lower PPDT in the perimenstrual phase and higher PPDT in the follicular and luteal phases of the menstrual cycle. Menstrual cycle could have been a confounding factor in this study if variations in the menstruation period existed between the two groups of subjects at the time of testing. Data was not collected on the menstrual cycle of women participating in this study.

Study Strengths

The present study has advantages over other studies measuring pressure pain detection threshold.[94,124] Previous pain threshold studies involving low back pain subjects have suggested that pain sensitivity measurements at a finger or ankle might adequately represent a subject's global pain threshold.[149,185] In these investigations the authors report that chronic low back pain sufferers tend to be generally less sensitive to pressure pain stimuli than control subjects. Unfortunately, pain threshold testing was only initiated at one unilateral distal site. The present investigation has advantages over previous studies as pain threshold was measured bilaterally at three sites distal to the low back region and three sites related anatomically to the lumbar spine. This study clearly provides a better overall perspective on global pain threshold than previous investigations. This study also recognized and accounted for the potentially

confounding effects of handedness and unilateral pain by measuring pain threshold bilaterally at all test sites. Unlike the present investigation, earlier pain threshold analyses did not control for the potentially confounding influences of medication, gender, or disability insurance involvement on pressure pain detection threshold.[149,212]

This investigation involved the use of a hand-held electronic pressure threshold meter. A 1 cm^2 rounded blunt tip at the end of the pressure threshold meter was applied to the subject's skin and pressure was slowly applied until the subject indicated the PPDT. This objective measure of PPDT was similar to a subjective measure of PPDT as applied in the clinical setting using a thumb or a finger. This type of pressure threshold meter has advantages over other types of pain threshold measurement such as the Forigone-Barbar pressure stimulator, thermal pain threshold measurement, electrical pain threshold measurement, ischemic pain threshold measurement, or ice immersion pain threshold measurement. Pain threshold measurement with the electronic pressure algometer is clinically relevant, elicits less fear in the subject, and simple to apply.

Chronic low back pain and pain free groups were fundamentally similar in this study. Demographic similarities were present in terms of age (41.52 and 42.17 years) and gender. Pain free and chronic low back pain subjects met stringent inclusion criteria and differed fundamentally on the basis pain and disability.

Conclusions

The purpose of this investigation was to compare the pressure pain detection threshold of chronic low back pain sufferers and healthy pain free volunteers. Pressure pain detection threshold measurement, with a pressure threshold meter, is an effective technique for quantifying pain sensitivity levels in persons with and without pain. In this study, the global pressure pain detection threshold of chronic low back pain sufferers was significantly less than that of pain free subjects. Chronic low back pain subjects also demonstrated a significant decrease in pressure pain detection threshold at test sites anatomically related to the lumbar spine (#3, #4, #5). Test sites distal to the low back and neuroanatomically unrelated to the lumbar spine (#1, #2, #5) were compared between chronic low back pain sufferers and pain free subjects and were not significantly different. All test sites demonstrated a trend towards lower pressure pain detection threshold measurements in the chronic low back pain group as compared to the pain free group. Hypersensitivity demonstrated in the low back region of chronic low back pain sufferers may be related to peripheral sensitization and increased reactivity of the primary afferent system, but psychosocial influences cannot be ruled out. Global hypersensitivity shown in chronic low back pain sufferers may be a product of the complex interplay between central sensitization and biopsychosocial factors. This study did not reveal a significant correlation between pain threshold and disability. Further research is needed to confirm the development of global hypersensitivity in persons experiencing persistent, ongoing low back pain.

Recommendations

As a result of this research, a number of recommendations are proposed:

1. This study demonstrated a definite trend towards a decrease in pressure pain detection threshold at all test sites. However, the differences in PPDT at sites unrelated to the lumbar spine were not statistically significantly lower in 'Chronic Low Back Pain' and 'Pain Free' groups. Further research might compare punctuate mechanical stimulation with blunt pressure to determine whether differences in pain threshold exist in relation to Aβ or C fiber stimulation.

2. Further research is required to determine a standardized protocol for pressure pain detection threshold measurement. This should include an investigation of the rate of pressure application, time lapse between repeated measures, and standardization of test sites for each body region.

3. The development of a pressure threshold meter capable of applying a constant rate of pressure is required for accurate PPDT measurement. A measurement tool of this nature should also allow subjects to engage a switch when the PPDT is reached.

4. The difference in PPDT between acute and chronic low back pain sufferers was not established in this research study. The question "Is there a difference in global and regional PPDT between chronic and acute low back pain sufferers?" remains unanswered. This study was not able to determine whether or not alterations in PPDT occur at sites unrelated to the lumbar spine in acute compared to chronic low back pain sufferers. Further research in this area is required. However, the issues surrounding difficulties in recruitment of acute low back pain sufferers may require further investigation.

Appendix A

Sample Size Calculation

Previous investigations have demonstrated the following mean and standard deviation values for pressure pain detection threshold:

(1) Healthy controls (points measured throughout all body regions)[160]:

- $\mu_1 = 5.45$ kg/cm^2
- $\sigma_1 = 2.75$ kg/cm^2

(2) Experimental groups with chronic and acute pain conditions (points measured throughout all body regions)[93,94,95,120 186]

- $\mu_2 = 3.29$ kg/cm^2
- $\sigma_2 = 1.374$ kg/cm^2

Sample size calculation for this study applies the following formula[213]:

$$n = 3 (PI * \sigma_p / \mu_1-\mu_2)^2$$

where:

n = number of subjects required per group

PI = Power index - calculated at .05 α and .10β

σ_p = pooled standard deviation values from previous studies

$= \sigma_1 + \sigma_2 / 2$

$= 1.374 + 2.75 / 2$

$= 2.062$ kg/cm^2

μ_1 = mean pressure detection threshold for healthy controls from previous studies

μ_2 = mean pressure detection threshold for painful subjects from previous studies

$n = 3 (PI * \sigma_p / \mu_1-\mu_2)^2 = 3 (3.24*2.062/ 2.16)2$

$= 28.7$ subjects/group

This value will be rounded up to 30 subjects / group for a total of 90 subjects in the study.

Appendix B
Consent Form

Part 1: Researcher Information		
Principal Investigator: Jason Giesbrecht Affiliation: University of Alberta, Department of Physical Therapy Contact Information: (780) 875-2651		
Name of Co-Investigator/Supervisor: Michele Crites Battié, PhD Affiliation: University of Alberta, Department of Physical Therapy Contact Information: (780) 492-5869		
Part 2: Consent of Subject		
	Yes	**No**
Do you understand that you have been asked to be in a research study?		
Have you read and received a copy of the attached information sheet?		
Do you understand the benefits and risks involved in taking part in this research study?		
Have you had an opportunity to ask questions and discuss the study?		
Do you understand that you are free to refuse to participate or withdraw from the study at any time? You do not have to give a reason and it will not affect your care.		
Has the issue of confidentiality been explained to you? Do you understand who will have access to your records/information?		
Part 3: Signatures		
This study was explained to me by: _____ Date: _____		
I agree to take part in this study. Signature of Research Participant: _____ Printed Name: _____		
Witness (if available): _____ Printed Name: _____ Subject # _____		
I believe that the person signing this form understands what is involved in the study and voluntarily agrees to participate. Researcher: _____ Printed Name: _____		

Appendix C

Letter of Request for Clinician Support

December 8, 2001

To: Physicians, Physical Therapists and Chiropractors
Lloydminster, AB/SK

Dear _____ (personalized),

I am a Physical Therapist with the Lloydminster Health District and a graduate student in the Faculty of Rehabilitation Medicine at the University of Alberta. I am planning to conduct an experiment in the Lloydminster area on pressure pain detection threshold changes in low back pain patients. The purpose of the research project is to identify any changes in the distribution and/or intensity of pressure pain detection threshold in acute and chronic low back pain sufferers compared to healthy pain free subjects. We intend to measure the pressure pain detection thresholds in acute and chronic low back pain sufferers and compare their pain detection threshold levels to one another and healthy pain free subjects. Alterations in the distribution or magnitude of sensory responses to mechanical stimulation may be explained through theories of sensitization and the biopsychosocial model of low back pain. The study results also may be applied clinically to the interpretation of assessment findings in patients with low back pain.

I am requesting the support and assistance of Lloydminster physicians, physical therapists and chiropractors in recruiting acute and chronic low back pain patients to participate in this clinical experiment. I will make further contact with you to discuss this project in more detail.

I appreciate your support and look forward to working with you in this endeavor to investigate low back pain.

Sincerely,

Jason Giesbrecht B.A., B.Sc.P.T.
Physical Therapist

Appendix D

Subject Questionnaire

1. What is your age (in years): _____

2. How long have you experienced back and/or leg pain (this episode)?
_____ days OR _____ months

3. Where is your pain? ☐ right side ☐ left side ☐ center ☐ both sides

4. Do you have leg pain below the knee? ☐ yes ☐ no, if yes ☐ right ☐ left

5. Do you currently use medication to relieve low back and/or leg pain? ☐ yes ☐ no

If yes, what kinds
_____

6. How has your employment status changed since you developed low back or leg pain?

(Please check one)
☐ currently off work because of low back pain.
☐ currently on modified duties at the same job because of low back pain.
☐ changed job (new position) because of low back pain.
☐ low back pain has not affected my employment status or ability to work.

Other,
explain:_____
_____
___
_____
___

Subject #: _____

Appendix E

Information Letter

Dear Participant,

You have been invited to take part in a research study. The study is titled "**Pressure Pain Detection Thresholds in Low Back Pain Patients and Pain Free Volunteers**".

The research team is listed below:

1. **Primary Investigator:** Jason Giesbrecht, Graduate Student, Department of Physical Therapy, University of Alberta, (306) 820-6055.
2. **Co-Investigator:** Michele Crites Battié PhD, Professor, Department of Physical Therapy, University of Alberta, (780) 492-5968.
3. **Research Assistant**: Christine Wilson, Physical Therapy Assistant.

The purpose of this project is to improve the physical assessment of persons suffering from low back pain. Your doctor or therapist will often assess an injured area by applying pressure to a sore spot during your physical exam. We would like to see how sensitive you are to light pressure. This study will examine your pressure pain threshold. This is the point at which pressure becomes painful. It is not a test of pain endurance. The test will stop at the first sign of pain. You will feel very little pain during the test.

Subjects will be asked about their present level of pain and how it affects their every day life. You have the right to refuse to answer any of the questions we ask. Any information you provide will be completely confidential (or private), except when professional codes of ethics or legislation (or the law) require reporting. The information you provide will be kept for at least five years after the study is done. The information will be kept in a secure area (i.e. locked filing cabinet). Your name or any other identifying information will not be attached to the information you provide. Instead a subject number will be assigned and used. Your name will also never be used in any presentations or publications of the study results. The information gathered for this study may be looked at again in the future to help us answer other study questions. If so, the ethics board will first review the study to ensure the information is used ethically.

The examiner will provide a review of the testing procedure. Also, a quick "test run" will be given on your thigh to make you familiar with the method of testing. Ample opportunity will be given for you to ask questions about the test or the study. You will then be asked to lie on your stomach or side on the treatment table and testing will begin. If you cannot tolerate either position, please discuss this with the examiner. She will help chose a more comfortable position for you.

Pressure threshold testing will involve light increasing pressure from a small metal tip (about the size of a pea). Pressure will be applied to both sides of your body. A total of 12 points on the legs, arms and trunk will be tested. You will be asked to identify your pain threshold, by saying "pain", when you first feel pain. **This is not a test of pain tolerance** but rather a test to see when pain begins. Each point will be tested 3 times. If you want to stop the test, for any reason, please notify the examiner and the test will be stopped. Stopping the test will not negatively affect you in any way. The entire testing procedure should take about 30 minutes. A small gift of $10 will be provided to thank you for taking part in this study. The results of this research project will be reported in a written report and a verbal presentation.

The risks of this test are minimal. Every possible step has been taken to avoid harm. Potential risks might include increased pain, bruising, or skin irritation. Any negative physical effects from testing will be managed with first aid procedures. If you have decided to stop taking anti-inflammatory medication (example: Naproxen) or over the counter pain medication (example: Tylenol, Advil, Ibuprofen) you may feel an increase in pain until you take the medication again. The benefits of this study may not affect you directly. Yet, the results of the study will improve our understanding of low back pain and assist with the assessment of low back pain patients.

Please contact Paul Hagler, Associate Dean of Research and Graduate Studies (780 492-9674), to report concerns or complaints. For information regarding ethical issues in this research project please contact Karen Turpin at (780) 492-0839.

Sincere thanks for your assistance in this study.

Initials: Researcher_____ Participant_____

Appendix F

Bioethics Review Committee Letter

Bioethics Committee
Lloydminster Health District
Lloydminster, SK/AB

March 7, 2002

To: Bioethics Committee,

I am a physical therapist with the Lloydminster Health District and also a Master's Candidate in the Department of Physical Therapy at the University of Alberta. At the advice of members of the Strategic Management Team I am providing a brief description of my intended investigation for your approval. Additional detailed information can be provided upon request.

The present study is designed to investigate the pressure pain detection threshold levels in acute and chronic low back pain patients compared to healthy control subjects. Ninety subjects (30 per group) will have their pressure pain detection thresholds measured by a trained examiner using a pressure threshold meter at 6 test sites on the right and left sides, throughout their body. The pressure pain detection threshold is defined as the lowest possible stimulus that can be perceived as pain. Tissue damage does not accompany pressure threshold testing.

A number of ethical issues have been considered in the design of this experiment. Informed consent will be obtained from each participant prior to the onset of testing. Detailed information will be provided to subjects by the project coordinator and will include a description of the testing procedure, a clear definition of pain detection threshold, the method to indicate pain detection threshold, expected discomforts, and risks associated (none anticipated) with testing and the right to refuse continued testing. Each subject will be instructed to discontinue participation in the study if she feels uncomfortable with the testing procedure. Each participant will be given a pressure test trial run on their right thigh to provide familiarity with the pressure threshold meter and allow the subject to ask questions about the testing procedure.

To ensure utmost safety for all subjects, all test points will be in areas of soft tissue away from open wounds or intensely inflamed tissue. Subjects will be provided with pillows or towels to maximize comfort in the testing position. If the subject cannot tolerate the prone position for any length of time, she will be allowed to roll onto side lying or return to an upright position at any point in the testing process and relieve her discomfort. Numbers will identify the data for each individual without a name attached to the data collection sheets or files to ensure anonymity. All

information provided by the subject and gathered during testing will remain confidential and anonymous.

The University of Alberta Health Ethics Review Board and my faculty advisory committee at the University of Alberta have approved this study.

I hope this information has been helpful. Please do not hesitate to contact me if further information is desired.

Sincerely,

Jason Giesbrecht B.A., B.Sc.PT.

Appendix G

Budget

The following budget outlines the tentative financial requirements for the experiment described above.

Item	Cost ($)
Pressure threshold meter	$1500
Subject remuneration	$900
Statistical Software	$150
Office Supplies, copying, etc	$100
Research Assistant Fees	$500
total	**$3150**

Appendix H

Roland Morris Disability Questionnaire

When your back hurts, you may find it difficult to do some of the things you normally do.

This list contains some sentences that people have used to describe themselves when they have back pain. When you read them, you may find that some stand out because they describe you today. As you read the list, think of yourself today. When you read a sentence that describes you today, put a tick against it. If the sentence does not describe you, then leave the space blank and go on to the next one. Remember; only tick the sentence if you are sure that it describes you today.

Because of my back or leg pain (sciatica) today:

Yes	No	
		1. I stay at home most of the time because of my back.
		2. I change position frequently to try to get my back comfortable.
		3. I walk more slowly than usual because of my back.
		4. Because of my back I am not doing any of the jobs that I usually do around the house
		5. Because of my back, I use a handrail to get upstairs.
		6. Because of my back, I lie down to rest more often.
		7. Because of my back, I have to hold on to something to get out of an easy chair.
		8. Because of my back, I try to get other people to do things for me.
		9. I get dressed more slowly than usual because of my back.
		10. I only stand up for short periods of time because of my back.
		11. Because of my back, I try not to bend or kneel down.
		12. I find it difficult to get out of a chair because of my back.
		13. My back is painful almost all the time.
		14. I find it difficult to turn over in bed because of my back.
		15. My appetite is not very good because of my back pain.
		16. I have trouble putting on my socks (or stockings) because of the pain in my back.
		17. I only walk short distances because of my back pain.
		18. I sleep less well because of my back.
		19. Because of my back pain, I get dressed with help form someone else.
		20. I sit down for most of the day because of my back.
		21. I avoid heavy jobs around the house because of my back.
		22. Because of my back pain, I am more irritable and back tempered with people than usual.
		23. Because of my back, I go upstairs more slowly than usual.
		24. I stay in bed most of the time because of my back.

Subject # _____

Appendix I

Subject Handout

Low Back Pain Research Project

Sponsored by the Lloydminster Health District

The Lloydminster Health District and the University of Alberta are involved in a **Low Back Pain Research Project.** This project is designed to improve the physical assessment of persons suffering from low back pain.

Subject Requirements

- Female,
- 20 – 60 years of age.
- NOT involved in a WCB or disability claim.
- Have suffered from low back pain for less than 3 weeks or more than 6 months.
- Have not taken anti-convulsant, anti-depressant, opioid analgesic, or benzodiazepine medication within 3 weeks of testing (please consult your doctor). Will not take anti-inflammatory medication or over the counter pain medication (example: Advil, Tylenol, Ibuprofen) within 24 hours prior to testing,
- Have not had low back surgery within six months of testing.
- Have not had physical treatment (i.e. physical therapy, chiropractic) 24 hours prior to testing.
- Must to able to tolerate lying on the stomach or the side for short periods of time.

Each subject will be asked to attend a 30-minute test session at the Lloydminster Hospital Physical Therapy Department. The exam will involve a simple test of your response to light pressure. You will receive a reward of $10 for taking part in the project. If you are interested or would like to know more about this project please call the number below or leave your name and number and you will be contacted.

Jason Giesbrecht PT

Graduate Student, University of Alberta, Department of Physical Therapy

(306) 820-6055 (work)

Please tear off here, give bottom portion to receptionist and keep top portion.

Name:
Phone Number:

☐ Yes, I want to be contacted to hear more about this research study.

Appendix J

Data Collection Sheet

Subject #: _____

	Cervical (1)	Forearm (2)	L3 (3)	L5 (4)	Calf (5)	PIP-D3 (6)
Right						
Left						

- Numbers in parenthesis indicate the number assigned to a specific test site for randomization.
- All values are measured in lbs/cm2 on the algometer.
- All values for right and left sides are the mean of three measurements at each specific point. The mean is automatically calculated by the electronic algometer.

Appendix K

Clinician Screening Checklist

Low Back Pain Research Project

University of Alberta, Faculty of Rehabilitation Medicine, Dept. of Physical Therapy

Inclusion Criteria for Low Back Pain Patients

Low Back Pain Patients

- Female,
- 20 – 60 years old,
- Not suffering from any other painful conditions,
- Not involved in a WCB or disability insurance claim,
- Have not taken opioid analgesic, anticonvulsant, antidepressant, or benzodiazepine medication for 3 weeks,
- Have not taken NSAIDs within 24 hours of testing.

AND

Acute low back and/or leg pain for less than **3 weeks**.

OR

Chronic low back pain and/or leg pain for more than **6 months**.

Subjects will be asked not to take NSAIDs 24 hours prior to testing

---

Please contact Jason Giesbrecht with questions or concerns at 820-6055.

Appendix L

NUMERIC RATING SCALE

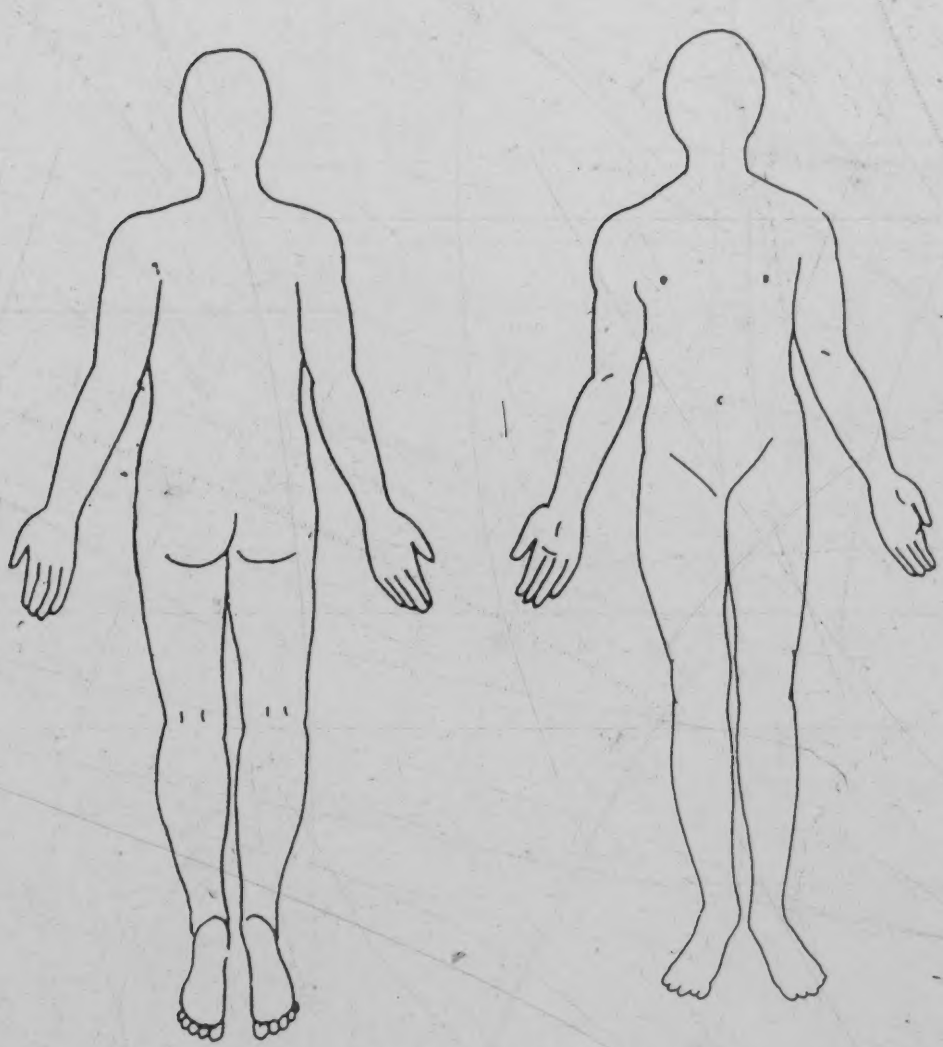
Instructions: Please indicate your current pain intensity circling one of the numbers provided below.

NO PAIN									WORST POSSIBLE PAIN
1	2	3	4	5	6	7	8	9	10

Subject # _____

Appendix M

Pain Drawing



Subject # _____

Appendix N

Certificate of Calibration - Pressure Threshold Meter

Certificate of Calibration

This device was factory calibrated to design specifications using weights traceable to the National Institute of Standards and Technology

ZEVEX QUALITY 14

TECH MEDICAL
a ZEVEX company
4314 ZEVEX Park Lane, SLC, UT 84123

Technician

8/9/01
Date

Appendix O

Advertisement for Pain Free Subjects

Volunteers Needed for Low Back Pain Study!

A study on low back pain, sponsored by the *University of Alberta and the Lloydminster Health District*, is being conducted at the Lloydminster Hospital. Pain free volunteers are needed to assist with this project.

Volunteers must be:
Female
Pain free
20-60 years old

All volunteers will receive $10 for their participation.
If interested please contact:
Jason Giesbrecht, Physical Therapy Department, 820-6055

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